VOLUME 58 NUMBER 4

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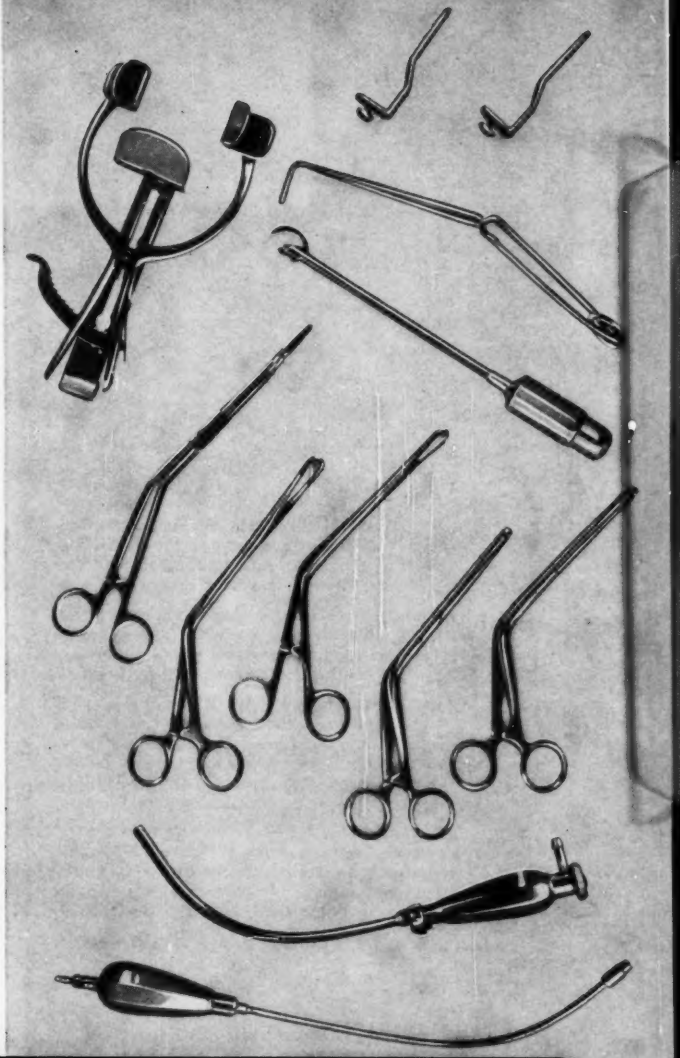

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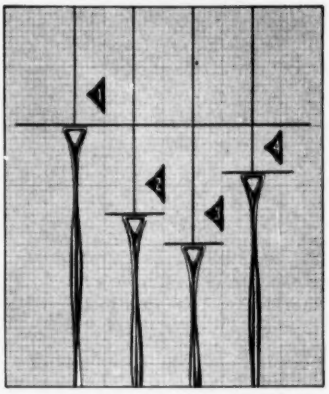

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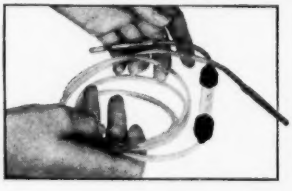

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HIATUS HERNIA

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AND
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HIATUS hernia is a far more frequent condition than has been generally accepted. It is not a rare thing. It is often overlooked, and the patients are treated for peptic ulcers, disease of the gallbladder or coronary heart disease. Of course, the same patient may have any of these conditions along with a hiatus hernia.

The allotted time will not permit a complete discussion of this subject, so we are confining it to a review of 24 cases which we have encountered in the last eighteen months at the Salt Lake Clinic. Incidentally, this is a larger number than was found in the previous thirty years. Our experience is probably not unique but goes to prove that once physicians are alerted to the existence of this type of hernia, it will be found much more frequently.

A thorough history will indicate definite symptoms suggestive of a hiatus hernia to the physician who is conscious of the existence of this condition. It occurs most frequently after 50 years of age, and reports have shown it to be more frequent in females than in males.

The commonest symptom is pain, usually under the lower half of the sternum or in the epigastrium. It may be referred to the back or up into the left shoulder and arm. It may be excruciating or described as a lump under the lower part of the sternum, pressure or a burning sensation. It occurs most frequently after heavy meals, especially if followed by stooping, lifting, lying down or rising up in bed or by anything that increases intra-abdominal pressure. The duration of the pain will depend on whether it is a sliding type of hernia or a fixed one or if it is complicated by an ulcer in the pouch, in which case it will be similar to the characteristic pain of any peptic ulcer.

Relief of pain on assumption of the upright position is characteristic of the sliding type of hernia. Dysphagia may be the first symptom experienced by the patient. Hot or cold foods or liquids, as well as roughage, may cause painful swallowing.

Read before the section on Gastro-Enterology and Proctology at the Ninety-Seventh Annual Session of the American Medical Association, Chicago, June 23, 1948.

Symptoms referable to the heart are common and lead to the diagnosis of heart disease. Hemorrhage occurs in hernias complicated by peptic ulcer in the pouch or from an associated ulcer in the stomach or duodenum. Symptoms referable to the other conditions occasionally associated with hiatus hernia may overshadow those referable to the hernia itself. The hernia is frequently overlooked until the symptoms of the associated condition are removed, as in excision of a gallbladder with stones, and one has to continue the search for a cause of the persisting pain.

A correct diagnosis can be made only by a roentgenologic examination. Some roentgenologists prefer the supine position at the time barium is swallowed, with rotation of the patient from side to side. We have been more successful when the Trendelenburg position has

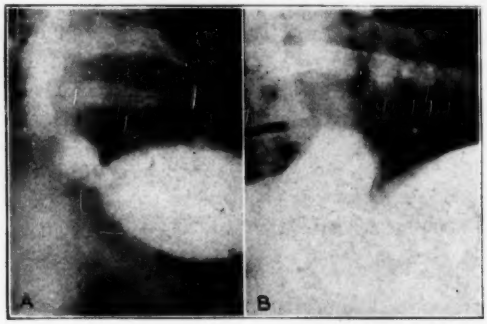

Fig. 1.—Hiatus hernia in case 1 (*A*) and case 2 (*B*).

been used. Generally the hernia is overlooked if the entire examination is made in the upright position, even though the patient be later examined in the supine position.

The following is a review of a few of our cases, with further emphasis on some of the characteristic symptoms and signs of hiatus hernia.

Case 1.—Mr. S. L. H., a farmer aged 40, complained of epigastric pain for one month. The patient was cranking a tractor and strained himself and had sudden gnawing epigastric pain. He was taken to a hospital for observation. Two conventional series of gastrointestinal roentgenograms were normal. He was released after several days, on a liquid diet. He continued to have intermittent pain.

Physical examination showed the patient to be stocky, with a weight of 177 pounds (80.3 Kg.). The blood pressure was 130 systolic and 70 diastolic. The roentgenogram of the gallbladder was normal, and one of the upper part of the gastrointestinal tract showed hiatus hernia (fig. 1).

Treatment.—He was placed on a reduction diet.

CASE 2.—Mr. L. L. W., an automobile mechanic aged 47, complained of burning pain near the right sternal border produced by pulling on a wrench when he was in a cramped position, present for two years. The pain occurred at times when he lay down. This man had been under treatment for coronary insufficiency for two years without relief of symptoms.

The patient was obese, with a weight of 182½ pounds (83 Kg.). The blood pressure was 146 systolic and 88 diastolic. He was otherwise normal. The electrocardiogram was normal, as were the stomach and duodenum. A roentgenogram of the upper part of the gastrointestinal tract revealed hiatus hernia (fig. 1 *B*).

Treatment.—He was placed on a reduction diet.

CASE 3.—Mr. F. C. B., aged 39, was a postmaster. His chief complaint was substernal pain, brought on while swallowing food and relieved by waiting or

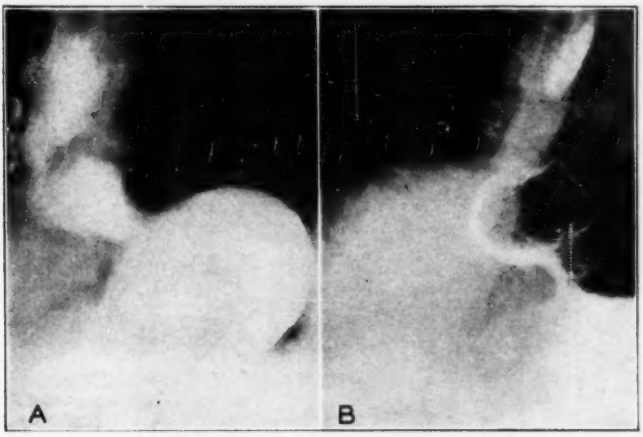

Fig. 2.—*A*, hiatus hernia in case 3; *B*, hiatus hernia partially filled with gas bubble in case 4.

by swallowing water. He also complained of heartburn, made worse by belching. He was rejected for military service on the basis of gastric neurosis. A conventional series of gastrointestinal roentgenograms at that time was reported to be normal. The patient was stocky, weighing 185 pounds (83 Kg.). The blood pressure was 120 systolic and 72 diastolic. There was a small indirect inguinal hernia on the left. Gastric analysis showed free hydrochloric acid amounting to 103 and total hydrochloric acid 114. The roentgenogram of the gallbladder was normal. A roentgenogram of the upper part of the gastrointestinal tract revealed hiatus hernia (fig. 2 *A*). The stomach and duodenum were normal.

Treatment.—The patient was placed on small bland feedings with antacids and advised to reduce his weight.

CASE 4.—Mr. W. M. W., aged 54, weighed 170 pounds (77 Kg.). On Feb. 7, 1942, he was awakened about 3 a. m. with excruciating pain in the epigastrium about eight hours after eating popcorn. At 6 a. m. he was seen by a

physician, who suspected a coronary occlusion. Intravenous administration of aminophylline gave no relief. Morphine, 0.5 grain (0.03 Gm.), was required for relief. The pain returned in six hours, again requiring morphine. Physical examination was noncontributory. An electrocardiogram and a roentgenogram of the gastrointestinal tract, made in the upright position, were normal. The genitourinary tract and the gallbladder were normal on roentgenograms, except that the gallbladder was slow in emptying.

The patient had no recurrence of pain until January 1948. After eating breakfast he stooped over from a standing position to tie his shoe and experienced a severe pain in the epigastrium and shortness of breath. Both subsided after he got into his car. One hour later, while walking briskly in the cold air, he was

Fig. 3.—Hiatus hernia in case 5.

seized with a pain in his chest and left arm. The pain subsided after he sat down. He continued to have the same pain each time he climbed stairs or walked in the open. The company physician took an electrocardiogram and a roentgenogram of his chest, which were normal.

Physical examination was essentially noncontributory. Laboratory examination revealed nothing abnormal except for a hemoglobin content of 17.15 Gm. and 5,790,000 red blood cells. An electrocardiogram showed posterior myocardial infarction. A roentgenogram of the gastrointestinal tract in the Trendelenburg position revealed a sliding type of hiatus hernia (fig. 2 *B*).

Treatment.—The patient was treated for the myocardial infarction and for overweight, with satisfactory results to date.

Case 5.—Mr. J. L. Y., aged 47, was a manufacturer. On Oct. 20, 1947, his chief complaint was pain beneath the xiphoid for three months. The onset

of epigastric pain had occurred three months previously, and it had lasted almost steadily for two days, with no associated symptoms. Since the onset the pain had returned with exertion and when the patient lay flat in bed. It was relieved by rest. The patient was obese, with a weight of 218 pounds (99 Kg.). The blood pressure was 120 systolic and 78 diastolic. Otherwise the condition was normal on physical examination. The roentgenogram of the gallbladder was normal. A roentgenogram of the upper part of the gastrointestinal tract revealed a hiatus hernia (fig. 3). The electrocardiogram was normal (fig. 4 *A*). The problem was to differentiate epigastric pain caused by a hiatus hernia in an obese man from that of coronary insufficiency.

An exercise test was done, which resulted in an abnormal electrocardiogram and brought on epigastric pain which was not relieved by oxygen and vasodilators (fig. 4 *B*). Later cardiograms were typical of anteroseptal infarct. The patient was hospitalized and treated for a myocardial infarction and given a reduction diet. Recurrence of epigastric pain was frequent unless he slept in a semireclining posture.

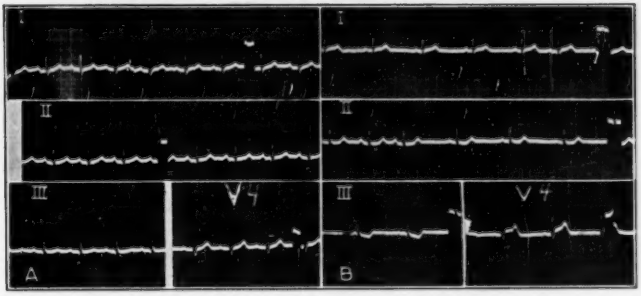

Fig. 4 (case 5).—*A*, electrocardiogram before exercise; *B*, electrocardiogram after exercise, showing definite evidence of coronary insufficiency.

He was last seen on May 20, 1948, had lost 27 pounds (12 Kg.) and was entirely free of pain.

Case 6.—Mrs. H. L. B., a housewife aged 68, weight 158 pounds (71.7 Kg.), underwent partial thyroidectomy for toxic goiter in 1928. She gained 20 pounds (9 Kg.) in the next two years and required thyroid extract for hypothyroidism.

In 1931 she was subjected to dilation and curettement and received radium for fibroid uterus. In 1932 she began to have pain in the lower part of the chest and epigastric pain, referred to both arms, which was relieved by nitroglycerin. This pain was associated with exertion. She continued to take nitroglycerin several times a day with relief as a rule, up to 1937, when she realized she had another type of pain, thought to be due to gallstones. The pain was severe in the lower right region of the chest and the epigastrium. It occurred during sleep or when the patient was getting up, lasting several hours. A roentgenogram of the gallbladder was normal. She had similar attacks of pain for ten years.

On Feb. 7, 1947, a roentgenogram of the gallbladder was normal. A roentgenogram of the gastrointestinal tract in the Trendelenburg position revealed a hiatus

hernia (fig. 5 *A*). The stomach and duodenum were normal. There was diverticulosis of the colon.

This patient undoubtedly had both a hiatus hernia and angina pectoris although numerous cardiograms taken over the last fourteen years have all been entirely normal.

She was placed on a reduction diet and advised to avoid exertion, especially after meals.

Case 7.—Mr. E. E. W., a salesman aged 53, on Sept. 19, 1947, presented the chief complaint of pain in the chest for nine years. He was kicked in the abdomen in 1920 and had vague epigastric pain intermittently until 1939, when he had severe epigastric and substernal pain which was diagnosed "myocardial infarction." He was hospitalized for three months and then stayed in bed nine more months. The pain continued intermittently while the patient was at bed rest and afterward. He was incapacitated because of "anginal syndrome."

The weight was 160 pounds (72.6 Kg.). The blood pressure was 136 systolic and 84 diastolic. He was of stocky build. Physical examination revealed a normal condition.

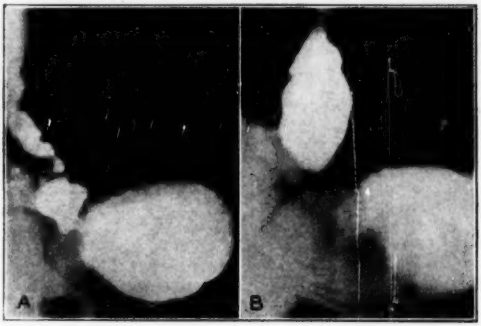

Fig. 5.—*A*, hiatus hernia in case 6; *B*, hiatus hernia for which the left phrenic nerve was crushed in case 7.

The roentgenogram of the chest was normal, as was the electrocardiogram after the exercise of stair climbing. The anoxia test showed vasomotor collapse at nine minutes but no electrocardiographic changes. A roentgenogram showed hiatus hernia (fig. 5 *B*).

Treatment.—A reduction diet of bland foods and administration of antispasmodics and alkalis gave no results. The patient asked for surgical treatment. Block of the phrenic nerve (left) with procaine hydrochloride relieved the pain; hence, the phrenic nerve was crushed, which gave complete relief, according to the patient when he was last seen.

Case 8.—Mrs. S. B. S., aged 52, weight 167 pounds (75.7 Kg.), on Jan. 27, 1948, presented the chief complaint of (1) pain in the lower part of the chest and the epigastrium for two months, made worse by turning over or rising up in bed and especially severe when she got up in the bathtub; (2) pain in the right arm and the side of neck, made worse by any motion of the head or arm and with no relation to epigastric pain, and (3) palpitation and dyspnea on exertion.

The physical examination revealed nothing abnormal except for a painful right arm and shoulder and tenderness of the brachial plexus. There was some limitation of motion of the neck. There was no scalenus anticus syndrome. A roentgenogram revealed arthritis of the cervical region of the spine and a hiatus hernia (fig. 6). The patient was put on a reduction diet, with small meals.

By Feb. 18, 1948, she had lost 14 pounds (6.4 Kg.) but the pain in the epigastrium had been constant. Procaine hydrochloride was injected into the left phrenic nerve, with complete relief of pain, and then the nerve was crushed.

She was seen at the clinic for follow-up on May 27, 1948, and had lost 13 pounds (6.4 Kg.) more and was entirely free of pain in spite of the fact that the diaphragm on the left side was again beginning to move with respiration.

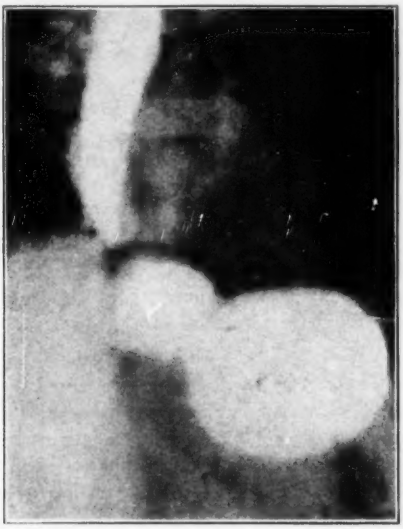

Fig. 6 (case 8).—Hiatus hernia for which the left phrenic nerve was crushed.

SUMMARY

Twenty-four cases of hiatus hernia were observed at the Salt Lake Clinic in the last eighteen months. This was more than were found in the previous thirty years.

Age.—The age varied between 33 and 68 years. Three patients were under 45 years of age.

Sex.—Seventeen patients were men, and 7 were women.

Weight.—Sixteen of the 24 patients were overweight.

Symptoms.—Twenty-three patients complained of either epigastric or substernal pain; 7 of these had radiation of the pain into the left

shoulder or arm. Only 1 had dysphagia. Two patients had gastrointestinal hemorrhages with anemia. The duration of symptoms was from six months to twenty years. The average duration was five and one-third years.

Form of Previous Therapy.—Seven patients had been treated for coronary heart disease, 5 had been treated for disease of the gallbladder, 7 had been treated for peptic ulcer and 7 had received no definite form of treatment. (Four patients had received treatment for both disease of the gallbladder and peptic ulcer or disease of the gallbladder and coronary heart disease.)

CONCLUSIONS

In conclusion, we wish to point out that whereas the frequency of hiatus hernia in recent medical literature varies from 0.6 per cent to as high as over 12 per cent in routine roentgenologic studies of the gastrointestinal tract it must be looked for by special technic if it is to be found more than occasionally. Hiatus hernia must be kept in mind in any case of atypical angina pectoris, duodenal ulcer, dysphagia, disease of the gallbladder, anemia from gastrointestinal bleeding or obscure upper abdominal pain or pain in the lower part of the chest which might be associated with increased intra-abdominal pressure. The index of suspicion has to be accompanied with a special technic of fluoroscopy if the true incidence of this disorder is ever to be appreciated.

INCIDENCE OF HIATUS HERNIA AND ASSOCIATED LESIONS DIAGNOSED BY ROENTGEN RAY

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WASHINGTON, D. C.

THE TRANSITION of hernia of the esophageal hiatus, more commonly called hiatus hernia, from a rare entity to one of comparative frequency has taken place in the past two decades. It was some years after the advent of diagnostic roentgenology that appreciation of the diagnostic value of changes in position of patients during barium study of the gastrointestinal tract led to routine study of the patient in the prone, supine or Trendelenburg positions. Ritvo[1] in 1930 emphasized the importance of this maneuver in bringing to light many previously undiagnosed hiatal hernias, particularly of the recurrent, small variety which may be seen only in the Trendelenburg position and may, indeed, be absent at another examination. Jenkinson and Roberts[2] estimated that only 5 per cent of hiatus hernias could be diagnosed if the erect position alone were used during examination.

Since the radiologist has attained a high degree of awareness of this entity, with proficiency in diagnosis, an evaluation of the frequency of hiatus hernia, diagnosed by the roentgen ray, was undertaken. The clinical significance of the presence of a hiatus hernia in a patient is largely determined by the clinician. Since the clinical manifestations presented by the patient with a hiatus hernia notoriously simulate those of other common entities diagnosed by the roentgen ray, it was thought to be of some value to determine the type and frequency of accompanying lesions in such patients. It was believed that such a study might add to clarification of the clinical status of the patient with a hiatus hernia.

Read before the Section on Gastro-Enterology and Proctology at the Ninety-Seventh Annual Session of the American Medical Association, Chicago, June 23, 1948.

From the Fifth and Sixth Medical Services (Boston University) and the Radiology Department, Boston City Hospital, Boston, and the Department of Medicine, Georgetown University School of Medicine, Washington, D. C.

1. Ritvo, M.: Hernia of the Stomach Through the Esophageal Orifice of the Diaphragm, J. A. M. A. **94**:15 (Jan. 4) 1930.

2. Jenkinson, E. L., and Roberts, E. W.: Lesions of the Diaphragm, Am. J. Roentgenol. **38**:584 (Oct.) 1937.

MATERIAL AND METHODS

The reports of all barium studies of the upper gastrointestinal tract done in the Radiology Department in two years, 1945 and 1946, at the Boston City Hospital were analyzed. Patients were referred from the various medical, surgical, specialty and outpatient services of a large general hospital mainly by house officers at the intern and resident level. In many cases gastrointestinal complaints were present, but in others it is likely that the roentgenologic study was part of a diagnostic work-up. Routinely, in this roentgen ray department study of the patient lying down or in the Trendelenburg position is done. No differentiation was made in the types of esophageal hernia since the clinical manifestations do not differ in the various types.

Table 1 reveals that among 3,448 patients examined in the two year period there were found 308 hernias of the esophageal hiatus, an incidence of 8.93 per cent. This was the second most frequent lesion

TABLE 1.—*Incidence of Most Frequent Diagnosis in Roentgen Examinations of the Upper Gastrointestinal Tract*

		Duodenal Ulcer		Hiatus Hernia		Gastric Ulcer		Carcinoma of Stomach	
	No. of Patients	No. of Cases	%	No. of Cases	%	No. of Cases	%	No. of Cases	%
1945.....	1,619	315	19.45	140	8.64	50	3.08	41	2.53
1946.....	1,829	390	21.32	168	9.18	70	3.82	60	3.28
Totals.....	3,448	705	20.41	308	8.93	120	3.48	101	2.92

diagnosed, duodenal ulcer having been found in 20.41 per cent of patients. It was more than twice as frequent as gastric ulcer or gastric carcinoma.

Examination of table 1 shows a good degree of consistency in the frequency of diagnosis of the lesions listed in both years. While the frequency of diagnosis in this series is higher than in most previously reported, some authors have also found a similar incidence. Schnepp [3] collected from the literature a thorough tabulation of the frequency of this lesion, finding an incidence varying from 0.02 to 8.0 per cent, which reflects its comparatively recent widespread recognition.

Harrington,[4] at the Mayo Clinic, estimated that its diagnosis was twenty times commoner in the past decade than in the previous two decades. Kirklin and Hodgson [5] recently reported 306 cases of esopha-

3. Schnepp, K. H.: Diaphragmatic Hernia: Its Incidence and Treatment, Illinois M. J. **83**:404 (June) 1943.

4. Harrington, S. W.: Diagnosis and Treatment of Various Types of Diaphragmatic Hernia, Am. J. Surg. **50**:381 (Nov.) 1940.

5. Kirklin, B. R., and Hodgson, J. R.: Roentgenologic Characteristics of Diaphragmatic Hernia, Am. J. Roentgenol. **58**:77 (July) 1947.

geal hiatus hernia diagnosed by roentgen ray in a one year period. Nuzum [6] reported an incidence of 12.27 per cent in 957 patients who had roentgenologic study in which a search for hiatus hernia was a routine part of the procedure.

Interesting, also, is Harrington's observation [4] on examination of the esophageal hiatus routinely in the course of 1,000 consecutive abdominal operations. He found that in 55 per cent of the cases the hiatus closely approximated the lower end of the esophagus. In 35 per cent one finger could be inserted between the esophagus and the margins of the esophageal hiatus; in 8 per cent two fingers could be inserted and in 2 per cent two to three fingers. In these last two groups herniation was thought to be potentially possible. These findings suggest an anatomic counterpart of closely comparative frequency to the incidence diagnosed by roentgen ray in this study.

Table 2 shows the incidence of the 308 hiatus hernias by sex and age. As in other series, women predominate, but by a much smaller margin than is usually given. For instance, Kirklin and Hodgson [5]

TABLE 2.—*Incidence of 308 Hiatus Hernias by Sex and Age*

Age	Women	Men	Totals	% by Decade	
20 to 30	1	2	3	0.97	
30 to 40	6	11	17	5.51	
40 to 50	21	16	37	12.01	
50 to 60	46	43	89	28.89	
60 to 70	41	35	76	24.67	76.61
70 to 80	41	30	71	23.05	
80 to 90	9	5	14	4.54	
Over 90	..	1	1	0.32	
	165	143	308		

reported the occurrence of hiatus hernias to be twice as frequent in women. Only 20 of the patients who had hiatus hernias were below 40 years of age. As in other series, the majority of patients are in the sixth to eighth decades of life. In the present study, such age groups accounted for 76.61 per cent of the cases. That the occurrence in the older age groups is not accounted for on the basis of the presence of a disproportion of elderly persons in this series is attested to by the frequency of diagnosis in this study of duodenal ulcer, a disease not restricted to the elderly.

The uniformity of age undoubtedly is of etiologic importance. Degenerative changes resulting in a loss of elasticity of the hiatal tissues contribute to the formation of the hernia. Abnormal stress, such as would occur in frequent episodes of coughing, retching or vomiting, could also stretch and possibly tear the tissues of the hiatal orifice. Increased intra-abdominal pressure from any cause is also

6. Nuzum, F. R.: Hernia of Esophageal Hiatus: Its Relationship to Angina Pectoris, Am. Heart J. **33**:724 (May) 1947.

a frequent factor in inducing changes at the esophageal hiatus. Obesity, large abdominal tumors or ascites may be contributing factors in this regard. Childbearing has a definite relationship to the development of hiatus hernia. Among 195 women in the third trimester of pregnancy, Rigler and Eneboe [7] found hiatal hernias in 18 per cent of 116 multiparas and 5 per cent of 79 primiparas. In only 3 of 10 women examined one to eighteen months post partum was the hernia again found. Schnepp [8] and Evans and Bouslog [9] have found hiatus hernia to explain some cases of "intractable heartburn" of pregnancy.

Many authors have proposed that hiatus hernia is primarily a congenital defect. In cases in which it occurs in infancy and usually involves the appearance of a greater part of the stomach, usually with other viscera, in the thoracic cavity, it is a congenital lesion. This is probably true also when it appears in adults in whom there is a short esophagus, with over a third of the stomach intrathoracically situated. Kirklin and Hodgson [5] stated that congenitally short esophagus with thoracic stomach is a rare lesion. It is really not a hiatus hernia since, with a congenitally short esophagus, the stomach has not herniated through the esophageal hiatus because it never was below the diaphragm at any time. From a roentgenologic point of view, they point out that three types of hiatus hernia may be differentiated. The commonest type, occurring in 66.5 per cent of their group, is the esophageal hiatal hernia in which the esophagogastric junction is above the diaphragm and both the stomach and the lower end of the esophagus are contained in the hernia. The next type is that in which there is a shortened esophagus, which occurred in 26 per cent of their cases, and it implies that at one time the esophagus was longer but that it has now become shortened. The third type, which occurred in 7.5 per cent of the cases, is the paraesophageal hernia in which a portion of the stomach herniates through the esophageal hiatus but the esophagogastric junction remains below the diaphragm and no esophagus is present in the hernia. In the present study, no differentiation is made between types since the clinical manifestations of all types are similar and do not lend themselves to differentiation. In the majority of cases, it seems rather unlikely for a congenital lesion to appear so frequently at the advanced ages at which hiatus hernia is found.

In this study 223 lesions were described by the radiologist as small, 68 as moderate and only 17 as large (over a third of the stomach in

7. Rigler, L. G., and Eneboe, J. B.: The Incidence of Hiatus Hernia in Pregnant Women and Its Significance, J. Thoracic Surg. **4**:262 (Feb.) 1935.

8. Schnepp, K. H.: Diaphragmatic Hernia as a Cause of "Intractable Heartburn" of Pregnancy, Am. J. Obst. & Gynec. **46**:142 (July) 1943.

9. Evans, J. R., and Bouslog, J. S.: Intractable Heartburn of Pregnancy, Radiology **34**:530 (May) 1940.

the thorax). Thirty-nine of the 308 lesions were described as being of the shortened esophagus type.

ASSOCIATED GASTROINTESTINAL LESIONS

Table 3 presents a study of the frequency and nature of associated conditions diagnosed concurrently with hiatus hernia. The associated diagnoses are not included in table 1 in the calculation of the incidence of duodenal ulcer, gastric ulcer or carcinoma. Thus, 25 per cent of this group of hiatus hernias are accompanied with conditions which in themselves may be responsible for, or potentially causative factors of, the clinical picture. The occurrence of 31 duodenal ulcers is an incidence rate of 10 per cent in the cases of hiatus hernia, as contrasted to 20.41 per cent in the whole group. There is obviously no etiologic significance.

TABLE 3.—*Associated Gastrointestinal Lesions Diagnosed by Roentgen Ray Study in 308 Cases of Hiatus Hernia*

	No. of Cases
Esophageal diverticulum	5
Esophageal varices	3
Esophagitis	3
Esophageal carcinoma	1
Gastric ulcer	2
Gastric carcinoma	4 (2) *
Gastric diverticulum	1
Gastritis, hypertrophic	7
Hypertrophy of pylorus	1
Duodenitis	3
Duodenal ulcer	31
Duodenal diverticulum	15
Jejunal diverticulum	3
Total	79

* Clinical investigation of these 4 cases revealed that in only 2 was carcinoma actually present. In the other 2 cases (1 at operation and 1 at necropsy) carcinoma of the stomach was not present.

Additionally, there were 5 cases in which an ulcer niche or crater was noted within the herniated portion. Huber and Andreson [10] in 1942 were able to collect from the literature 33 cases of ulcer in situ and added 4 new ones. There have been several single case reports since that time.

Though there were no esophageal ulcers noted in this series, it is of interest that Dick and Hurst [11] were of the opinion that chronic esophageal ulcer rarely occurs without the presence of a hiatus hernia

10. Huber, F., and Andreson, L. H.: Gastric and Duodenal Ulcer Associated with Lesions of the Lower End of the Esophagus and the Cardia of the Stomach, Am. J. Roentgenol. **48**:158 (Aug.) 1942.

11. Dick, R. C. S., and Hurst, A.: Chronic Peptic Ulcer of the Esophagus and Its Association with Congenitally Short Esophagus and Diaphragmatic Hernia, Quart. J. Med. **11**:105 (April) 1942.

of the congenitally short esophagus type. Particularly with the short esophagus type, there is loss of the normal valvular mechanism at the cardia, which allows regurgitation of stomach contents into the esophagus, especially when the patient is lying down. The acid stomach content thus aids in formation of the esophageal ulcer.

Johnstone,[12] on the other hand also, felt that a hiatus hernia was invariably present, being found in 20 of his 21 cases of esophageal ulcer. However, he strongly dissented to the proposed idea that the shortened esophagus was of a congenital nature. Rather, the esophagus may become shortened because of fibrosis and healing of the ulcer; in other words, there is an acquired shortening of the esophagus. This better explains the old age of the groups involved than the postulation of a basic congenital lesion. Dick and Hurst objected to this on the basis that if sufficient cicatrization occurs to shorten the esophagus and pull the stomach into the thorax it might be expected to cause esophageal stenosis, which apparently had not occurred in their experience. Interesting on this point is Benedict's thorough study[13] of 44 cases of benign esophageal nontraumatic stricture, in which series a hiatus hernia was present in 17, or 38 per cent. Thus, hiatus hernia is seen to be an important factor in benign esophageal stenosis.

Wilkinson[14] reported 6 duodenal ulcers in 70 cases of hiatus hernia, while Polley[15] found 4 duodenal ulcers in 47 cases. Jankelson and Morein[16] stated that ulcer of the stomach and duodenum accompanied hiatus hernia in 25 per cent of the cases. The findings in the present series are fairly consistent with the other reports.

Weintraub and Tuggle[17] found that in 310 cases of diverticulum of the duodenum 9 per cent of the patients had an associated hiatus hernia. Conversely, Polley[15] found that in 10 per cent of 47 cases of hiatus hernia a duodenal diverticulum was present. This compares with an incidence of 5 per cent in this series. Diverticulosis of the colon occurred in 34 per cent of Polley's cases and in 23 per cent of Wilkinson's[14] 70 cases of hiatus hernia.

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12. Johnstone, A. S.: Peptic Ulceration of the Esophagus with Partial Thoracic Stomach, Brit. J. Radiol. **16**:357 (Dec.) 1943.

13. Benedict, E. B.: Benign Stricture of Esophagus, Gastroenterology **6**:328 (April) 1946.

14. Wilkinson, S. A.: Diaphragmatic Hernia, New England J. Med. **210**:1105 (May 24) 1934.

15. Polley, R. F.: Congenital Short Esophagus with Thoracic Stomach and Esophageal Hiatus Hernias, J. A. M. A. **116**:821 (March 1) 1941.

16. Jankelson, I. R., and Morein, S.: Complications of Diaphragmatic Hernia, Rev. Gastroenterol. **7**:134 (March-April) 1940.

17. Weintraub, S., and Tuggle, A.: Duodenal Diverticula, Radiology **36**:297 (March) 1941.

While not studied in the present series, it is interesting to note that either roentgenologic or histologic evidence of chronic cholecystitis and cholelithiasis occurred in 14 per cent of Wilkinson's cases, in 15 per cent of Polley's and in 17 per cent of Jones's.[18]

Carcinoma of the esophagus or stomach in combination with a hiatus hernia is uncommonly reported. In this series a midesophageal carcinoma in association with a hiatus hernia was the only malignant esophageal lesion. Whereas gastric carcinoma in combination with a hiatus hernia was diagnosed by roentgen ray in 4 cases, examination of the clinical records revealed that only 2 of the patients actually had carcinoma of the stomach. In the other 2 cases, in 1 operation and in 1 necropsy revealed that carcinoma was not present. In this series of patients with hiatus hernia there was, then, an incidence of gastric carcinoma of 0.65 per cent, as contrasted with an incidence of 3.48 per cent in the total number of patients studied. As with duodenal ulcer, this suggests that there may be some degree of protection afforded against the occurrence of carcinoma of the stomach by the presence of a hiatus hernia.

The foregoing data serve to indicate the degree of frequency with which associated gastrointestinal lesions occur in cases of hernia of the esophageal hiatus. The clinical importance of such findings is the point that must be repeatedly stressed on searching for and ruling out other gastrointestinal entities before symptoms are ascribed to a hiatus hernia. In a study of 50 patients with hiatus hernia, Radloff and King[19] found one or more concomitant diseases in 69 per cent, the correction of which often afforded symptomatic relief.

COMMENT

There is little doubt that hiatus hernias, regardless of size, may be completely asymptomatic and may be demonstrated accidentally. This is the case in many instances in which accompanying lesions cause the symptoms. However, it is difficult to determine from the literature exactly how frequently hernias occur without symptoms. Wilkinson[14] stated that in only 4 of his 70 cases were there no symptoms that could possibly be related to the hiatus hernia. Moersch[20] found that all but 19 of 246 patients with a hiatus hernia had symptoms directly attributable to the hernia. On the other hand, Root and Pritchett[21]

18. Jones, C. M.: Hiatal Esophageal Hernia, New England J. Med. **225**:963 (Dec. 18) 1941.

19. Radloff, F. F., and King, R. L.: Esophageal Hiatus Hernia, Gastroenterology **9**:249 (Sept.) 1947.

20. Moersch, H. J.: Hiatal Hernia, Ann. Otol., Rhin. & Laryng. **47**:754 (Sept.) 1938.

21. Root, J. C., and Pritchett, C. P.: Diaphragmatic Hernia, Cleveland Clin. Quart. **5**:203 (July) 1938.

stated that 19 of 31 patients with hiatus hernia had no symptoms referable to the hernia, but there were 14 associated gastrointestinal lesions in the asymptomatic group.

I have not been able to find in the literature a study of a sizable group of patients without gastrointestinal symptoms in the 50 to 70 age range in which roentgenologic examination for hiatus hernia specifically was undertaken. Such a report is required so that some idea of a control can be obtained to contrast with the available reports which are usually concerned with patients presenting symptoms of such a nature that roentgen examination of the gastrointestinal tract is indicated.

From the clinical point of view, the question of whether the presenting lesion is congenital or acquired is of no importance. The size of the hiatus hernia is also of little importance. This is stressed by Harrington [4] and Jones,[18] who pointed out that many of the symptoms occurred most frequently in patients with the smallest hernias.

TABLE 4.—*Distribution of Symptoms in 223 Patients (Modified from Jones [18] and Ohler and Ritvo [22])*

Symptoms	No. of Cases	%
Epigastric pain	129	58
Nausea, vomiting or regurgitation	119	53
Heartburn	38	17
Dysphagia	18	8
Dyspnea	28	12
Substernal pain	63	28
Shoulder pain	42	18
Arm or hand pain	13	6

Table 4, modified from Jones [18] and Ohler and Ritvo,[22] presents the predominating symptoms in 223 carefully studied patients. There may be various combinations of symptoms, but in many cases one type of symptomatology predominates. Ritvo [1] stated the belief that the most typical complaint was a feeling of weight or pressure under the xiphoid process, which comes on during or soon after meals and is relieved by taking a hot drink or by walking about for a few minutes. Jones,[18] in studying 45 patients with small hernias, found, as have others, that factors which may either precipitate or aggravate symptoms are exertion, emotional tension, the intake of excessive food and lying down. Somewhat typical is the tendency for symptoms to occur in attacks intermittently with periods of complete freedom. The physical examination is of little value in diagnosis. Methods of obtaining symptomatic relief were variable and included assumption of the upright position, vomiting, belching, the use of antacids and the use of atropine and nitroglycerin.

22. Ohler, W. R., and Ritvo, M.: Diaphragmatic (Hiatus) Hernia, New England J. Med. **229**:191 (July 29) 1943.

It is apparent that there is no pathognomonic pattern which the symptoms of hiatus hernia assume. Rather, its manifestations group themselves into syndromes, which may in an individual case simulate esophageal, gastric or duodenal, cholecystic and coronary artery disease as well as gastrointestinal bleeding and anemia of undetermined cause; this gives a good idea of the differential diagnostic problem.

In this study of roentgenologic findings in association with hiatus hernia, there was another diagnosis in 1 of every 4 cases. Clinical study would no doubt reveal that the hiatus hernia was a coincidental finding as regards the explanation of symptoms in some of these cases. On the other hand, the hiatus hernia should not be overlooked as a potential source of symptoms because another gastrointestinal lesion is present. Obviously, too, some of the associated lesions, such as duodenal and jejunal diverticula, are thought of as usually, but not always, being nonsymptomatic. There is no easy method of determining, in the presence of such combinations, which lesion is productive of the patient's symptoms, since either lesion alone may produce them. Observation and careful attention to the clinical course under treatment will usually uncover the answer to this perplexing problem.

SUMMARY

1. The relative frequency of hiatus hernia is indicated by presentation of 308 cases in which the diagnosis was made by roentgen ray in a two year period.

2. Hiatus hernia was the second commonest lesion diagnosed in this material by roentgenologic study of the upper part of the gastrointestinal tract. Duodenal ulcer was the most frequent diagnosis.

3. In 1 of every 4 cases of hiatus hernia there was roentgenologic diagnosis of an associated gastrointestinal lesion.

4. Careful evaluation of the clinical course is necessary to determine the part that hiatus hernia has in producing symptoms when an associated lesion is present.

DIFFERENTIAL DIAGNOSIS OF HIATUS HERNIA AND CORONARY ARTERY DISEASE

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IT IS THE purpose of this paper to emphasize the increasing incidence of hiatus hernia and coronary artery disease and to demonstrate that they may coexist, so that the presence of one condition does not exclude the other, and that by means of objective tests the presence of coronary disease can be ascertained and its relative importance properly evaluated. For those who wish to study the subject of hiatus hernia per se several earlier comprehensive reports should be consulted.[1]

Although many use the terms hiatus hernia and diaphragmatic hernia interchangeably, in this report we will survey only herniation of the stomach through the esophageal opening of the diaphragm and not rupture through the diaphragm itself. The latter is far less common than hiatus hernia.

The differential diagnosis of hiatus hernia and angina pectoris due to coronary disease is of paramount importance. The diagnosis of hiatus

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1. (*a*) Hedblom, C. A.: Diaphragmatic Hernia: A Study of Three Hundred and Seventy-Eight Cases in Which Operation Was Performed, J. A. M. A. **85**:947 (Sept. 26) 1925. (*b*) von Bergmann, G.: Das "epiphrenale Syndrom," seine Beziehung zur Angina pectoris und zum Kardiospasmus, Deutsche med. Wchnschr. **58**:605 (April 15) 1932. (*c*) Harrington, S. W., and Kirklin, B. R.: The Clinical and Roentgenologic Manifestations and Surgical Treatment of Diaphragmatic Hernia, with a Review of One Hundred and Thirty-One Cases, Radiology **30**:147 (Feb.) 1938. (*d*) Reid, W. D.: Hiatus Hernia Simulating Cardiac Infarction: Report of a Case, New England J. Med. **223**:50 (July 11) 1940. (*e*) Jones, C. M.: Hiatus Esophageal Hernia, with Special Reference to a Comparison of Its Symptoms with Those of Angina Pectoris, ibid. **225**:963 (Dec. 18) 1941. (*f*) Levy, M. D., and Duggan, L. B.: Hiatus Hernia of the Stomach: Incidence, Symptoms and Medical Management in 1,220 Gastro-Intestinal Cases, South. M. J. **34**:351 (April) 1941.

hernia has been made with increasing frequency.[2] Nuzum [2e] actually discovered 25 instances of hiatus hernia in 100 consecutive cases of angina pectoris. He then studied roentgenologically another group of 957 patients and found hiatus hernia in 78 cases. Levy and Duggan [1f] estimated that hiatus hernia was present in 2 to 3 per cent of all the cases in which roentgen study for gastrointestinal disease was made.

Coronary artery disease, too, is being detected with increasing frequency. Each annual United States census reports an increasing number of persons suffering from this illness. Master [3] reported that in this country a half to one million attacks of coronary occlusion alone occur every year. We [2d] have already emphasized the increasing incidence of both hiatus hernia and coronary disease. Actually hiatus hernia and coronary disease occur in the same age group, 40 to 70 years.[4]

It has long been known that hiatus hernia may produce pain in the chest which can simulate angina pectoris due to coronary disease.[5] Herman and Singer [5f] and Linn [6] even observed simulation of coronary occlusion. Conversely, it is well known that coronary artery disease may be associated with symptoms r erred to the gastrointestinal tract. Also,

2. (*a*) Cunha, F.: Recurrent "Hiatus Hernia" Syndrome of von Bergmann, Am. J. Digest. Dis. & Nutrition **1**:170 (May) 1934. (*b*) Moschowitz, E.: The Simultaneous Association of Hiatus Hernia and Coronary Disease, J. Mt. Sinai Hosp. **4**:272 (Nov.-Dec.) 1937. (*c*) Nuzum, R.: Hernia of Esophageal-Hiatus: Its Relationship to Angina Pectoris, Am. Heart J. **33**:724 (May) 1947. (*d*) Dack, S.; Stone, J.; Grishman, A., and Master, A. M.: Differential Diagnosis of Diaphragmatic Hernia and Coronary Heart Disease, Bull. New York Acad. Med. **24**:396 (June) 1948. (*e*) von Bergmann.[1b] (*f*) Harrington.[1c] (*g*) Levy and Duggan.[1f]

3. Master, A. M.: Incidence of Acute Coronary Occlusion, Am. Heart J. **33**:135 (Feb.) 1947.

4. Jones.[1e] Dack and others.[2d]

5. (*a*) Jackson, D. E., and Jackson, H. L.: Experimental and Clinical Observations Regarding Angina Pectoris and Some Related Symptoms, J. Lab. & Clin. Med. **21**:993 (July) 1936. (*b*) Cossio, P., and Fustinoni, O.: Angina de pecho y hernia diafragmatica, Rev. argent. de cardiol. **9**:217 (Sept.-Oct.) 1942. (*c*) Porter, W. B.: Cardio-Diaphragmatic Syndrome, Virginia M. Monthly **70**:179 (April) 1943. (*d*) Beck, W., and Huffman, J.: The Importance of Diaphragmatic Hernia in the Differential Diagnosis of Coronary Artery Disease, Stanford M. Bull. **3**:181 (Nov.) 1945. (*e*) Clark, W. E.: Gastrointestinal Conditions Simulating or Aggravating Cardiovascular Disease, J. A. M. A. **128**:352 (June 2) 1945. (*f*) Herman, M., and Singer, E.: Para-Esophageal Hiatal Hernia: A Case Manifesting Gastrointestinal and Cardiac Symptoms and Presenting Itself on X-Ray as a Mediastinal Tumor, New York State J. Med. **46**:1020 (May 1) 1946. (*g*) von Bergmann.[1b] (*h*) Harrington and Kirklin.[1c] Cunha.[2a] Dack and others.[2d]

6. Linn, G. C.: Hiatus Hernia Confused with Coronary Thrombosis, New York State J. Med. **48**:303 (Feb. 1) 1948.

the two diseases may coexist.[7] It is therefore obvious that the presence of one of these diseases does not exclude the other. To complicate the situation further, a patient may give no typical history of angina pectoris and yet suffer from coronary disease; and, on the other hand, in the absence of coronary disease a patient with hiatus hernia may present a typical anginal syndrome, with classic radiation of the pain to the left arm or the back, precipitated by effort, excitement or meals. Yet after operation on the hernia the symptoms have disappeared.[8] Hence the history of the patient alone cannot be utilized as a means of diagnosis of either hiatus hernia or coronary artery disease.

It must be remembered that even in the presence of normal coronary arteries there are certain complications of hiatus hernia that may precipitate acute coronary insufficiency.[9] The most important of these is hemorrhage from erosion or ulceration of the mucous membrane of the esophagus or the herniated stomach.[10] With the decrease in hemoglobin, anoxia of the heart muscle may occur, producing changes in the electrocardiogram and even infarction of the left ventricle. In addition to hemorrhage, other complications of hiatus hernia which may occasionally occur are intense nausea, vomiting and dysentery. The patient often goes into peripheral vascular shock and acute coronary insufficiency may develop.

We have dwelt on the fact that hiatus hernia and angina pectoris may coexist and that similar symptoms may appear in each disease. The following symptoms, although more common in cases of angina pectoris, are found in both conditions: epigastric or substernal pain or

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7. (*a*) Stubenbord, W. D.: Paroxysmal Auricular Fibrillation in Association with Hiatus Hernia: Report of a Case, Ann. Int. Med. **18**:406 (March) 1943. (*b*) Groedel, F. M.: Gastrointestinal Disorders Simulating Circulatory Disease and Vice Versa, Am. J. Digest. Dis. **12**:73 (March) 1945. (*c*) Radloff, F. F., and King, R. L.: Esophageal Hiatus Hernia, Gastroenterology **9**:249 (Sept.) 1947. Moschcowitz.[2b] Nuzum.[2c] Dack and others.[2d]

8. Jones, C. M., and Chapman, W. P.: Studies on the Mechanism of the Pain of Angina Pectoris with Particular Relation to Hiatus Hernia, Tr. A. Am. Physicians **57**:139, 1942. Herman and Singer.[5f] Linn.[6]

9. Master, A. M., and Jaffe, H. L.: (*a*) Coronary Insufficiency and Myocardial Necrosis Due to Acute Hemorrhage, J. Mt. Sinai Hosp. **7**:26 (May-June) 1940. (*b*) Master, A. M.; Gubner, R.; Dack, S., and Jaffe, H. L.: Differentiation of Acute Coronary Insufficiency with Myocardial Infarction from Coronary Occlusion, Arch. Int. Med. **67**:647 (March) 1941. (*c*) Master, A. M.; Jaffe, H. L.; Dack, S., and Grishman, A.: Coronary Occlusion, Coronary Insufficiency and Angina Pectoris, Am. Heart J. **27**:803 (June) 1944. (*d*) Master, A. M.; Dack, S.; Grishman, A.; Field, L. E., and Horn, H.: Acute Coronary Insufficiency: An Entity; Shock, Hemorrhage and Pulmonary Embolism as Factors in Its Production, J. Mt. Sinai Hosp. **14**:8 (May-June) 1947.

10. Bock, A. V.; Dulin, J. W., and Brooke, P. A.: Diaphragmatic Hernia and Secondary Anemia: Ten Cases, New England J. Med. **209**:615 and 624 (Sept. 28) 1933. Harrington and Kirklin.[1c]

distress radiating to the left shoulder, aggravated by exertion and food and relieved by belching and by the use of nitroglycerin; indigestion, vomiting and other gastric symptoms. Nitroglycerin and often other antispasmodics will relieve the pain of both lesions.

The epigastric or thoracic pain or distress developing in the recumbent position or when the patient is bent forward and relieved by standing is characteristic of hiatus hernia. Moreover, the pain or pressure of hiatus hernia is apt to radiate to the right, and nitroglycerin does not relieve it as readily as atropine, belladonna and other antispasmodics and sedatives.[1e] The relation of body position to the symptomatology of hiatus hernia is not consistent. We are reminded of a man of 63 with severe coronary sclerosis whose attacks of angina pectoris were often precipitated or aggravated in the prone or sitting position and relieved by standing up. He died of myocardial infarction due to coronary insufficiency; at autopsy no hiatus hernia was found but only the severe coronary disease. It is clear, then, that objective tests of coronary disease are essential in distinguishing hiatus hernia and coronary disease, and for this reason we have made liberal use of the standard two step exercise test [11] and, to a lesser extent, of the 10 per cent anoxemia test.[12]

It has been known for a long time that reflexes arising from the diaphragm, esophagus, stomach or gallbladder, normal or diseased, may produce cardiac signs and symptoms and affect the coronary circulation.[13]

11. (*a*) Master, A. M.; Friedman, R., and Dack, S.: The Electrocardiogram After Standard Exercise as a Functional Test of the Heart, Am. Heart J. **24**:777 (Dec.) 1942. (*b*) Master, A. M.; Dack, S., and Jaffe, H. L.: Cardiac Efficiency and Prognosis Following Recovery from Acute Coronary Occlusion: The Results of Various Functional Tests, J. A. M. A. **120**:1271 (Dec. 19) 1942. (*c*) Master, A. M.; Nuzie, C.; Brown, R. C., and Parker, R. C., Jr.: The Electrocardiagram and the "Two-Step" Exercise: A Test of Cardiac Function and Coronary Insufficiency, Am. J. M. Sc. **207**:435 (April) 1944.

12. Levy, R. L.; Williams, N. E.; Bruenn, H. G., and Carr, H. A.: The "Anoxemia Test" in the Diagnosis of Coronary Insufficiency, Am. Heart J. **21**:634 (May) 1941.

13. (*a*) Schrager, V. L., and Ivy, A. C.: Symptoms Produced by Distention of the Gallbladder and Biliary Ducts, Surg., Gynec. & Obst. **47**:1 (July) 1928. (*b*) Roemheld, L.: The Treatment of the Gastrocardiac Syndrome (Gastric Cardiopathy), Am. J. M. Sc. **182**:13 (July) 1931. (*c*) Scott, H. G., and Ivy, A. C.: Viscerocardiac Reflexes: An Experimental Study in Frogs and Dogs, Arch. Int. Med. **49**:227 (Feb.) 1932. (*d*) Dietrich S., and Schwiegk, K.: Das Schmerzproblem der Angina pectoris, Klin. Wchnschr. **12**:135 (Jan. 28) 1933. (*e*) Hinrichsen, J., and Ivy, A. C.: Effect of Stimulation of Visceral Nerves on Coronary Flow in Dogs, ibid. **51**:932 (June) 1933. (*f*) Weiss, S., and Ferris, E. B., Jr.: Adams-Stokes' Syndrome with Transient Complete Heart Block of Vagovagal Reflex Origin: Mechanism and Treatment, ibid. **54**:931 (Dec.) 1934. (*g*) Greene, C. W.: Control of the Coronary Blood Flow by Reflexes Arising in Widely Distributed Regions of the Body, Am. J. Physiol. **113**:399 (Oct.) 1935. (*h*) Gilbert, N. C.: Vasomotor Changes in the Coronary Arteries and Their

(Footnote continued on next page)

This may apply particularly to hiatus hernia. The thoracic, phrenic, vagus and spinal nerve pathways are much the same for the diaphragm, lower end of the esophagus and stomach.[14] It was thought that painful sensations arising from distention of the esophagus or herniated portion of the stomach or from the diaphragm were transmitted through these visceral afferent fibers to the upper thoracic spinal segments, from which they were referred to the precordium and the left arm. Miller [15] has presented detailed descriptions of these nerve pathways which explain the radiation of pain to the sternum, precordium and left arm in hiatus hernia as well as in other thoracic and abdominal visceral diseases. Afferent pathways from the stomach, diaphragm and esophagus "may enter practically all the dorsal spinal routes and through these multiple afferent fibers . . . cardio-aortic and abdominal visceral references are mediated . . . the pain of these abdominal and pelvic conditions is focused in the heart and its immediate neighborhood and even overflows into the brachial and cervical plexuses."

Since the physiologic experiments of Dietrich and Schweigk [13d] and Gilbert, Fenn and LeRoy,[13i] it has been assumed by some clinicians that hiatus hernia can produce pain in the chest by reflex vasoconstriction of the coronary arteries. The latter investigators observed diminution of coronary blood flow in dogs when the stomach was distended, a vagal reflex effect which could be abolished by vagotomy or atropinization. However, earlier experiments in dogs by Henrichson and Ivy [13e] had failed to demonstrate any significant decrease in coronary blood flow following stimulation of the intact vagus nerve below the heart or excitation or distention of the upper abdominal viscera.

In an attempt to elucidate this problem by further clinical investigation, we decided to use the two step exercise and anoxemia tests on patients with hiatus hernia. The intensive application of these function tests has been of immeasurable help in attaining a more precise differential diagnosis of angina pectoris due to coronary insufficiency and of thoracic pain of noncardiac origin.

Possible Significance, J. A. M. A. **113**:1925 (Nov. 25) 1939. (*i*) Gilbert, N. C.; Fenn, G. K., and LeRoy, G. V.: The Effect of Distention of Abdominal Viscera on the Coronary Blood Flow and on Angina Pectoris, ibid. **115**: 1962 (Dec. 7) 1940. (*j*) Morrison, L. M., and Swalm, W. A.: Role of the Gastrointestinal Tract in Production of Cardiac Symptoms: Experimental and Clinical Observations, ibid. **114**:217 (Jan. 20) 1940. (*k*) Brown, C. F. G., and Dolkart, R. E.: The Significance of Gastrointestinal Tract Abnormalities as Related to the Management of Cardiac Disorders, M. Clin. North America **28**:107 (Jan.) 1944. von Bergmann.[1b]

14. Jones.[1e] Jackson and Jackson.[5a] Porter.[5c] Linn.[6] Schrager and Ivy.[13a] Weiss and Ferris.[13f] Greene.[13g] Morrison and Swalm.[13j]

15. Miller, H. R.: (*a*) Angina Pectoris, Baltimore, Williams & Wilkins Company, 1939, pp. 132-136; (*b*) Central Autonomic Regulations in Health and Disease, New York, Grune & Stratton, Inc., 1942, p. 149.

MATERIAL AND METHODS

Fifty-seven patients with positive roentgenologic evidence of hiatus hernia were chosen for study. The majority of the patients were referred for roentgen examination because of their gastrointestinal symptoms, and the examinations were carried out in a routine fashion by the department of radiology.

The series comprised 31 men and 26 women, an almost equal distribution. The great majority were between 40 and 70 years of age; 3 were younger, and 2 were older.

To ascertain the presence or absence of organic heart disease the following clinical determinations were made: a complete history was obtained; physical, teleoroentgenographic and roentgenoscopic examinations were made; the resting electrocardiogram including augmented unipolar extremity and Wilson precordial leads was obtained, and the two step exercise electrocardiogram and the 10 per cent anoxemia test were made.

The criterion for abnormality of the standard two step or double two step electrocardiogram [11] was the appearance after the exercise of RS-T depressions of 1 mm. or more or inversion or flattening of an upright T wave. The standard test was performed in 28 of the 32 patients whose resting electrocardiogram was normal. In 13 of the patients whose standard two step electrocardiogram was normal the test was repeated with double the number of trips called for in the standard tables, i. e., the double two step test.

The anoxemia test was performed on 16 of the 32 patients whose resting electrocardiogram was normal. This test consists of breathing a mixture of 10 per cent oxygen and 90 per cent nitrogen for twenty minutes. Our criteria for a positive reaction to the test were those described by Levy and his co-workers.[12]

RESULTS

The clinical findings and the results of the function test are recorded in the table. From the detailed history the patients were divided into four groups, as follows: group I, comprising 15 patients in whom only gastrointestinal symptoms were present (cases 1 to 15); group II, comprising 29 patients (cases 16 to 44) with both gastrointestinal and cardiac complaints; group III, comprising 3 patients (cases 45 to 47) whose presenting symptoms were chiefly cardiovascular, and group IV, comprising 10 patients (cases 48 to 57) with significant anemia whose symptoms were due to the anemia and to gastrointestinal disturbances.

In group I the chief complaints were entirely gastrointestinal in nature, namely, epigastric distress or pain, indigestion, difficulty in swallowing, regurgitation of food, substernal fulness, heartburn, nausea and vomiting. The symptoms were generally related to meals and were relieved on standing up. The cardiac investigation revealed no evidence of organic cardiac disease in 12 of the 15 patients. In 3 patients, however, despite the absence of cardiac symptoms, the presence of heart disease was demonstrated by left ventricular enlargement in 2 (cases 13 and 14) and an abnormal electrocardiogram in 1 (case 15). It was our impression that the heart disease in these 3 patients was not associated with any of the symptoms. In 4 other

Group I

No.	Case	Age	Sex	Gastrointestinal Symptoms	Cardiac Symptoms
1	E. S.	60	M	Epigastric distress, heartburn, vomiting	.....
2	S. T.	59	M	Nausea, vomiting blood	.....
3	V. W.	55	M	Epigastric distress, nausea, vomiting	Substernal pain, not on effort
4	I. B.	52	F	Epigastric pain, improved on standing or walking	Pain in left side of chest
5	G. B.	56	M	Dysphagia, nausea, vomiting	.....
6	C. S.	57	F	Epigastric distress, indigestion	.....
7	T. M.	75	F	Indigestion, nausea	Dyspnea, heaviness in chest, not on effort; auricular premature beats, hypertension
8	R. E.	58	F	.....	Pain in left side of chest on leaning forward and after meals
9	S. B.	50	M	Indigestion	Pain in left shoulder
10	M. C.	50	M	Indigestion, epigastric distress referred to right upper quadrant and right side of chest, relieved on standing	Ventricular premature beats
11	E. G.	44	M	Epigastric distress	.....
12	S. N.	46	M	Duodenal ulcer	Pressure on lower end of sternum, worse lying down, not on effort
13	H. Y.	68	M	Epigastric dysphagia, nausea, vomiting, worse lying down	Dyspnea, enlarged left ventricle
14	M. I.	40	F	Epigastric distress, relieved on standing or walking	Substernal pain, relieved on standing or walking. Ventricular premature beats, slight left ventricular enlargement
15	S. G.	71	M	Indigestion, dysphagia, nausea, vomiting	Auricular premature beats

Group II

16	C. R.	49	M	Indigestion, vomiting, dysphagia	Precordial pressure, worse after effort and meals
17	J. C.	46	M	Epigastric distress	Typical angina pectoris on effort, treated for coronary occlusion
18	B. Z.	60	M	Epigastric distress, heartburn	Retrosternal pressure, mild on effort
19	D. F.	38	M	Epigastric distress, indigestion, nausea, vomiting	Angina pectoris on effort, dyspnea
20	J. B.	37	F	Epigastric distress; gallbladder disease	Angina pectoris on effort
21	H. S.	49	M	Indigestion	Angina pectoris
22	T. T.	66	F	Dysphagia, regurgitation, substernal fulness	Old coronary occlusion; angina pectoris on effort
23	R. F.	50	F	Epigastric distress	Dyspnea, hypertension, enlarged left ventricle
24	B. H.	63	F	Abdominal discomfort, belching	Hypertension; auricular premature beats
25	J. P.	39	M	Belching	Pressure in chest, relieved on standing and walking; old coronary occlusion
26	B. M.	39	M	Belching	Substernal pressure on effort
27	B. R.	68	M	Peptic ulcer symptoms	Dyspnea
28	M. S.	72	F	Epigastric distress	Precordial pain on exertion, substernal burning, hypertension
29	E. G.	72	F	Nausea, vomiting blood, substernal burning	Substernal pain referred to back; dyspnea on effort
30	C. M.	47	M	Epigastric discomfort referred to left shoulder, worse lying down	Sharp substernal pain; treated twice for coronary occlusion
31	C. V.	72	F	Epigastric distress	Dyspnea, chest pain referred to epigastrium; syncope
32	M. F.	53	M	Epigastric distress	Typical angina pectoris, ventricular premature beats; treated for coronary occlusion; hypertension
33	H. W.	70	F	Indigestion	Paroxysmal auricular fibrillation, left ventricular enlargement, premature beats
34	H. R.	71	M	Heartburn, indigestion	Typical severe constricting precordial pain relieved on standing; left ventricular enlargement
35	R. C.	61	F	Epigastric distress, indigestion and heartburn	Substernal pain not on effort, with heartburn, hypertension, ventricular premature beat
36	B. P.	46	F	Epigastric distress, constant nocturnal aggravation	Dyspnea—pain worse on excitement
37	B. P.	45	F	Epigastric distress, nausea, vomiting blood	Typical angina pectoris on effort, hypertension, coronary occlusion
38	M. N.	71	M	Epigastric distress, indigestion, nausea, vomiting	Substernal pain, dyspnea, left ventricular enlargement
39	M. S.	63	F	Epigastric distress	Left ventricular enlargement, paroxysmal tachycardia, hypertension, angina pectoris on effort
40	J. B.	52	M	Epigastric distress	Typical angina pectoris on effort
41	J. V.	50	M	Indigestion, heartburn	Typical angina pectoris on exertion
42	E. S.	55	F	Indigestion, nausea, vomiting	Angina pectoris on effort, mild; paroxysmal auricular fibrillation, hypertension
43	G. B.	50	M	Epigastric distress, indigestion	Acute coronary occlusion
44	A. C.	51	F	Indigestion, duodenal ulcer	Angina pectoris on effort

Hernia and Coronary Disease

Electrocardiogram	Exercise Test, Standard	Exercise Test, Double	10% Oxygen Test	Hemoglobin	Associated Conditions	Organic Heart Disease	Comment
—	—	..	—	—	.....	—	
—	—	..	..	79%	.....	—	
—	—	..	..	—	Referred substernal pain	—	P-R interval, 0.24 second
—	—	..	..	—	Duodenal ulcer; referred pain in left shoulder	—	
—	—	—	—	—	.....	—	
..	..	..	..	—	.....	—	
—	..	..	..	..	Cardiac irregularity	—	
—	—	—	—	—	Referred pain in left side of chest	—	
—	—	..	—	—	Referred pain in left shoulder	—	
—	—	..	..	—	Cardiac irregularity	—	
—	—	..	..	—	.....	—	
—	—	+	—	—	.....	—	
..	..	..	..	72%	.....	+	
..	..	..	..	75%	Substernal pain	+	
+	..	..	..	75%	Diverticulum of esophagus	+	
—	—	..	—	—	Neurocirculatory asthenia	—	
—	—	—	..	—	Typical angina pectoris	—	
—	—	..	—	—	Mild angina pectoris	—	
—	—	—	—	—	Pain in chest and left arm, bronchiectasis	—	
—	—	..	..	75%	Angina pectoris, gallbladder disease	—	
—	..	..	..	—	Angina pectoris	—	
+	..	..	..	73%	Coronary disease	+	Old coronary occlusion
+	..	..	..	—	Hypertension	+	Marked left axis deviation and high voltage electrocardiogram
+	..	..	..	..	Hypertension	+	Left axis deviation in electrocardiogram
+	..	..	..	..	Coronary disease	+	Old coronary occlusion
—	+	+	..	—	Typical angina pectoris	+	
+	..	..	..	75%	Ulcer and coronary disease	+	
+	..	..	..	—	Coronary disease	+	
+	..	..	..	77%	Coronary disease	+	
+	..	..	..	—	Cardiac disease; referred substernal pain	+	
+	..	..	..	—	.....	+	Sinoauricular block; gallbladder disease
+	—	+	—	—	Coronary disease	+	
+	..	..	..	—	Coronary disease	+	Esophageal diverticulosis
+	..	..	..	—	Acute coronary occlusion	+	
—	+	..	+	—	Coronary disease	+	
+	—	..	—	65 to 78%	Angina pectoris	+	
+	..	..	..	—	Coronary disease	+	
+	..	..	..	—	Coronary disease	+	
+	..	..	..	—	Hypertension and coronary disease	+	
+	..	..	..	—	Coronary disease	+	
—	—	+	..	..	Acute coronary insufficiency, with abnormal electrocardiogram	+	
—	±	..	..	—	Angina pectoris	+	
+	..	..	..	—	Coronary disease	+	
—	—	+	—	—	Coronary disease	+	

No.	Case	Age	Sex	Gastrointestinal Symptoms	Cardiac Symptoms
Group III					
45	A. L.	50	M	.....	Typical angina pectoris on effort
46	R. R.	61	F	.....	Typical angina pectoris on effort; auricular fibrillation
47	J. C.	50	M	.....	Pressure above heart, not related to effort or meals, relieved on standing; auricular premature beats
Group IV					
48	I. F.	49	M	Epigastric pains relieved by standing up	Pain in left side of chest, not on effort or meals
49	R. B.	56	F	Epigastric distress, dysphagia, nausea, vomiting	Precordial and left shoulder pain, dyspnea, weakness
50	N. F.	77	M	Epigastric distress, duodenal ulcer	Retrosternal burning
51	A. M.	52	M	Tarry stools	Precordial pain, not on effort; dyspnea, weakness
52	L. B.	46	F	Epigastric distress, nausea, vomiting, relieved on standing	Dyspnea, ventricular premature beats
53	M. B.	58	F	Nausea, vomiting	Precordial pain referred to left shoulder, dyspnea, hypertension
54	C. F.	40	F	.....	Substernal pressure on effort; dyspnea; left ventricular enlargement
55	P. B.	56	M	Heartburn, vomiting blood	Hypertension, left ventricular enlargement
56	R. L.	68	F	Epigastric distress, heartburn, nausea, vomiting blood	Dyspnea, cardiac murmur
57	M. L.	52	F	Epigastric distress, nausea, vomiting	Pain referred to left shoulder; dyspnea

patients epigastric pain was referred to the chest or the left shoulder, but no evidence of coronary disease was elicited. The referred thoracic pain was generally vague in nature and was not related to effort but usually occurred after eating.

The largest subdivision was group II (cases 16 to 44), in which both gastrointestinal and cardiac symptoms were manifested. In addition to the usual symptoms of a gastrointestinal disturbance, there was pain or pressure in the substernal or precordial region, occurring spontaneously with or after exertion, palpitation, dyspnea and tachycardia. For example, in case 16 the chief complaint was indigestion and frequent vomiting, and yet there was also precordial pressure that was distinctly increased by effort. In case 17 the epigastric distress was related definitely to effort; the patient had been treated previously for coronary occlusion. In case 18, although heartburn and epigastric distress were frequent they did occur after mild effort. Similarly, in case 19, in which the chief complaints were epigastric distress, nausea and vomiting, the epigastric pressure bore a close relationship to effort and there was also shortness of breath on exertion. In 23 of this group of 29 patients cardiac disease was demonstrated by the objective examinations. Thus, in cases 22 to 25 inclusive the resting electrocardiogram was abnormal and, as a matter of fact, 2 of the patients had suffered coronary occlusion. Similar abnormalities were discovered in the remaining cases in which organic heart disease was listed. In 3 cases (cases 26, 41 and 44) the positive two step electrocardiogram was the only objective sign of organic heart disease. In 1 instance

and Coronary Disease—Continued

Electrocardiogram	Exercise Test, Standard	Exercise Test, Double	10% Oxygen Test	Hemoglobin	Associated Conditions	Organic Heart Disease	Comment
+	+	+	..	—	Coronary disease	+	
+	..	..	..	—	Coronary disease	+	
—	+	+	—	—	Coronary disease, cardiac irregularity	+	
—	—	—	—	60%	Anemia, duodenal ulcer	—	
—	..	..	..	35%	Anemia and coronary insufficiency	—	
—	..	..	..	39%	Anemia and referred pain	—	
—	—	—	—	54%	Anemia and coronary insufficiency	—	
—	—	..	—	59%	Anemia	—	
+	..	..	..	27%	Severe anemia and coronary insufficiency	+	S-T depression in electrocardiogram
—	..	..	..	45%	Moderate anemia and coronary insufficiency	+	
+	..	..	..	45%	Anemia and coronary insufficiency	+	
+	..	..	..	28%	Severe anemia and coronary insufficiency	+	Gallbladder disease
+	..	..	..	57%	Anemia	+	

(case 35) the reaction to the 10 per cent oxygen test as well as to the two step test was positive.

In 6 patients (cases 16 to 21) whose symptoms were suggestive of cardiac disease the presence of the latter could not be confirmed by the objective tests or examinations. All 6 patients experienced retrosternal or precordial pressure or pain on effort in addition to their gastrointestinal complaints. Negative reactions to anoxemia and two step tests were obtained in 3 of these and negative reactions to two step tests in 2. The work-up was not considered complete in the remaining case, since these tests could not be performed. These 6 cases are of importance because organic coronary artery disease could not be demonstrated despite the history of a typical anginal syndrome on effort. Therefore, it is possible that in these cases the hiatus hernia was the only factor initiating the thoracic pain through the reflex pathways previously mentioned. We doubt whether reflex coronary vasoconstriction was an important precipitating factor, since no evidence of coronary insufficiency could be elicited by the function tests. It is more likely that the pain may have been referred from the esophagus or stomach to the thoracic wall without involvement of the coronary circulation.

There was a group of 3 cases (group III, cases 45, 46 and 47) in which only cardiac symptoms were present. The patient in case 45 had angina pectoris on effort; electrocardiograms at rest and after exercise were abnormal, but there were no gastrointestinal symptoms associated with hiatus hernia. The patient in case 46 had typical angina pectoris on effort, auricular fibrillation and an abnormal resting electrocardiogram.

The patient in case 47 experienced pressure over the heart which was unrelated to effort but was relieved by standing up. Premature beats were present, and the exercise electrocardiogram was abnormal. In none of these 3 patients would hiatus hernia have been suspected by history alone except perhaps in the last, in whom the thoracic pain was relieved in the upright position.

Group IV consisted of 10 patients (cases 48 to 57) who were anemic, the blood hemoglobin content ranging from 28 to 60 per cent of normal. The anemia was produced by chronic bleeding from the stomach. It generally was associated with symptoms referable to the cardiovascular system, such as dyspnea, weakness and precordial pain on effort, suggesting the presence of coronary insufficiency. In 5 patients (cases 53 to 57) the diagnosis of organic heart disease was established by the presence of abnormal electrocardiograms or cardiac enlargement. In 3 of the other 5 cases function tests were done and the results were negative, but they were performed after the anemia had disappeared. It is our impression that in all the patients of this group the anemia resulting from hemorrhage was the important factor in producing the cardiac symptoms, probably on the basis of coronary insufficiency. Correction of the anemia usually resulted in disappearance of the cardiac symptoms.

Cardiac irregularities were encountered in 14 patients. In order of frequency they were auricular premature beats in 5 patients, ventricular premature beats in 5, paroxysmal auricular fibrillation in 2, paroxysmal tachycardia in 1 and sinus arrest with nodal escape in 1. Two different arrhythmias were recorded in 2 patients (cases 33 and 42). All but 4 of the 14 patients were found to have other clinical evidence of organic heart disease in addition to the hiatus hernia. Nevertheless, it is possible that the hiatus hernia was important in initiating the arrhythmia. Reflex sinus arrest, heart block and auricular fibrillation have been observed in experimental distention of the esophagus [13j] and in cardiospasm [16] as a result of stimulation of, or pressure on, the vagus nerve. Stubenford [7a] reported paroxysmal auricular fibrillation in a case of hiatus hernia and suggested that a search be made for this lesion in every case of paroxysmal auricular fibrillation. It is possible that the auricular and ventricular premature beats in our cases were also reflexly produced by the hiatus hernia.

REPORT OF CASES

The following short histories are illustrative both of the type of patient we encountered and of the points of distinction between hiatus hernia and coronary artery disease.

16. Iglauer, S., and Schwartz, B. A.: Heart-Block Periodically Induced by the Swallowing of Food in a Patient with Cardiospasm (Vagovagal Syncope), Ann. Otol., Rhin. & Laryng. **45**:875 (Sept.) 1936. Weiss.[13b]

CASE 8 (group I).—R. E., a 58 year old woman, complained of dull pain in the lower left anterior region of the chest on bending forward and after eating for at least seven years. There was also pain in the left arm. Neither the pain in the chest nor that in the arm was ever related to exertion. Physical examination of the chest and abdomen revealed no abnormalities. Roentgenologic examination of the stomach revealed a moderately large hiatus hernia, with a good portion of the stomach situated in the thorax (fig. 1 *A*). The heart appeared normal in size and configuration. The electrocardiogram, including unipolar extremity and multiple precordial leads, was entirely normal. No significant change occurred after the standard and double two step exercise tests and the anoxemia test (fig. 1 *B*). Pain in the chest did not appear during any of these function tests.

It is reasonable to expect that had the thoracic pain been due to coronary disease, evidence of it would have become manifest in seven years. Furthermore, the negative findings in the two step and anoxemia tests proved absence of coronary disease. The aggravation of the complaints on bending forward is characteristic of hiatus hernia.

CASE 41 (group II).—J. V., a man of 50 years, had experienced indigestion and heartburn for the past twenty years and precordial pain on effort during the past four months. Physical examination of the chest and abdomen was noncontributory. Roentgenologic examination revealed a short esophagus, with herniation of the stomach into the thorax (fig. 2 *A*). The resting electrocardiogram was normal, but the double two step exercise test produced distinct depression of the RS-T segment in the standard and precordial leads, characteristic of coronary insufficiency on effort (fig. 2 *B*).

It is apparent that in this patient the long-standing history of dyspepsia was associated with the hiatus hernia, but an anginal syndrome had develôped recently on the basis of coronary artery sclerosis, a lesion unrelated to the hernia. The two step exercise electrocardiogram was essential for the diagnosis of coronary disease.

CASE 34 (group II).—H. R., a man of 71 years, presented a two week history of indigestion and heartburn relieved by alkali and bismuth powders. One week previously he began to experience precordial and substernal pain radiating through to the back, occurring spontaneously and also on walking. The pain was worse when lying down and was relieved by standing up. Since the character of the symptoms suggested the presence of hiatus hernia, roentgen examination of the gastrointestinal tract was carried out; this diagnosis was confirmed (fig. 3 *A*). However, on the day of this examination a severe attack of constricting precordial pain developed. Severe precordial pain recurred during the next few days and was associated with the classic clinical signs of acute coronary occlusion with myocardial infarction, which was confirmed by serial electrocardiograms (fig. 3 *B*) specific for this disease.

The clinical course in this case suggests that the patient was first seen during the premonitory phase of acute coronary occlusion. The resting electrocardiogram was pathognomonic of coronary occlusion.

From a practical point of view, it should be emphasized that in an elderly patient the manifestations of both hiatus hernia and severe coronary artery disease may develop simultaneously. The mere demonstration of the presence of hiatus hernia should not lead one to attribute all the symptoms to this one lesion.

CASE 17 (group II).—J. C., a 46 year old man, for twenty years experienced frequent heart attacks which were diagnosed as angina pectoris due to coronary disease. The attacks were unrelated to activity and consisted of substernal pain

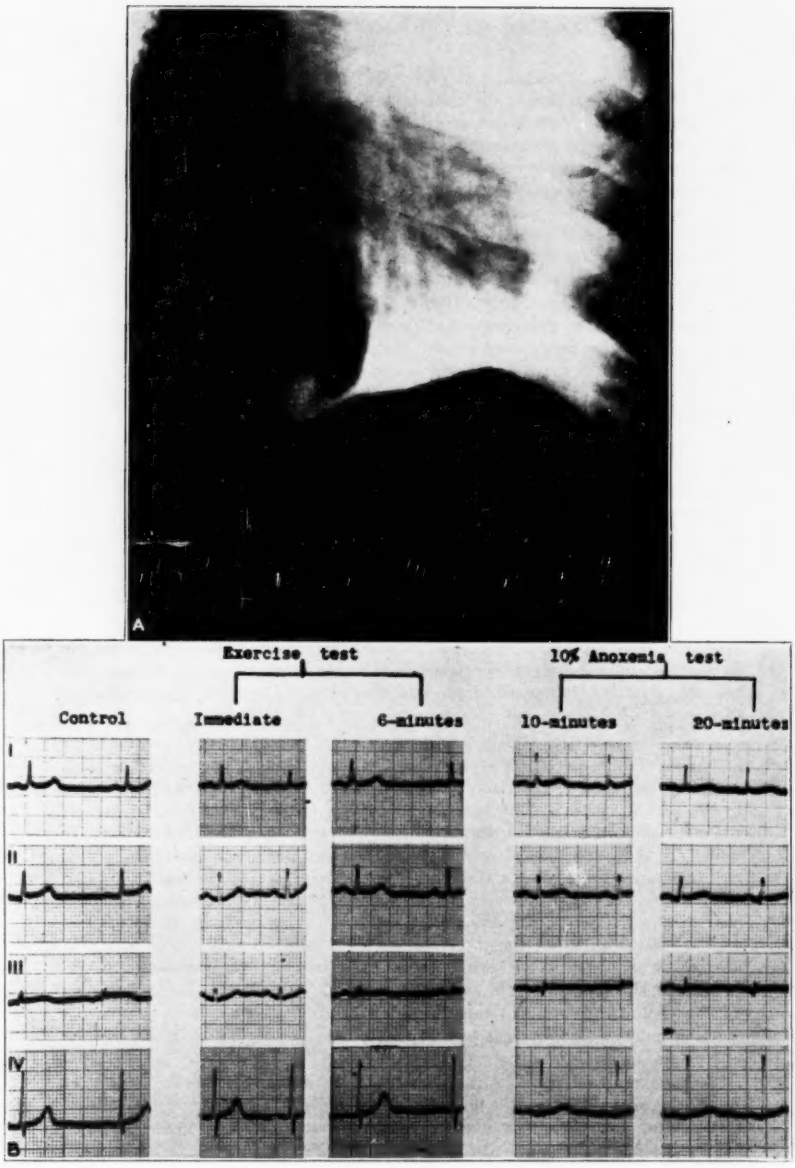

Fig. 1 (case 8).—*A*, moderately large hiatus hernia without organic heart disease. Dull pain in lower anterior region of chest after eating, but not related to exertion. No objective evidence of heart disease. *B*, normal electrocardiogram at rest and after standard two step and 10 per cent anoxemia tests.

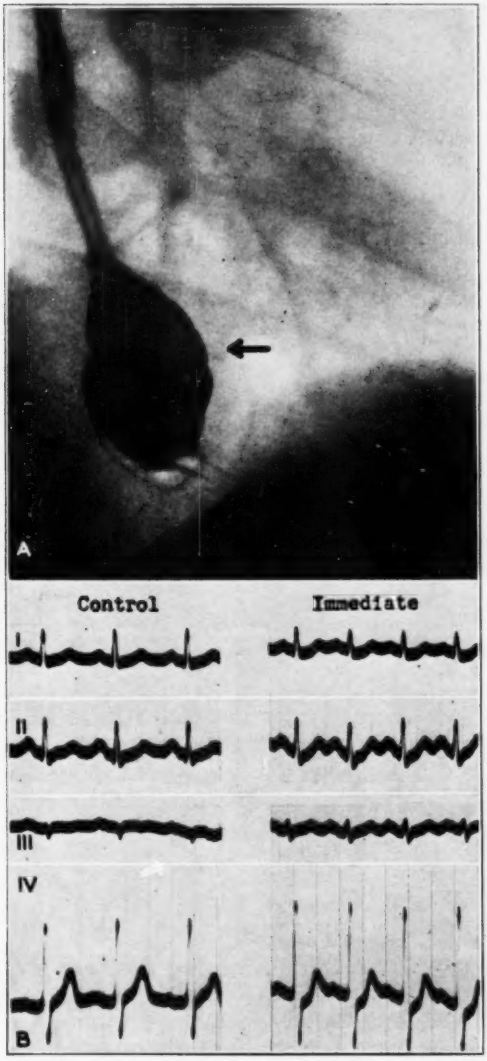

Fig. 2 (case 41).—*A*, small hiatus hernia and coronary artery disease. Indigestion for twenty years; precordial pain on effort for four months. *B*, control electrocardiogram normal. Positive reaction to standard two step exercise test (depression of RS-T segment), especially in precordial lead, indicating coronary insufficiency on effort due to coronary sclerosis.

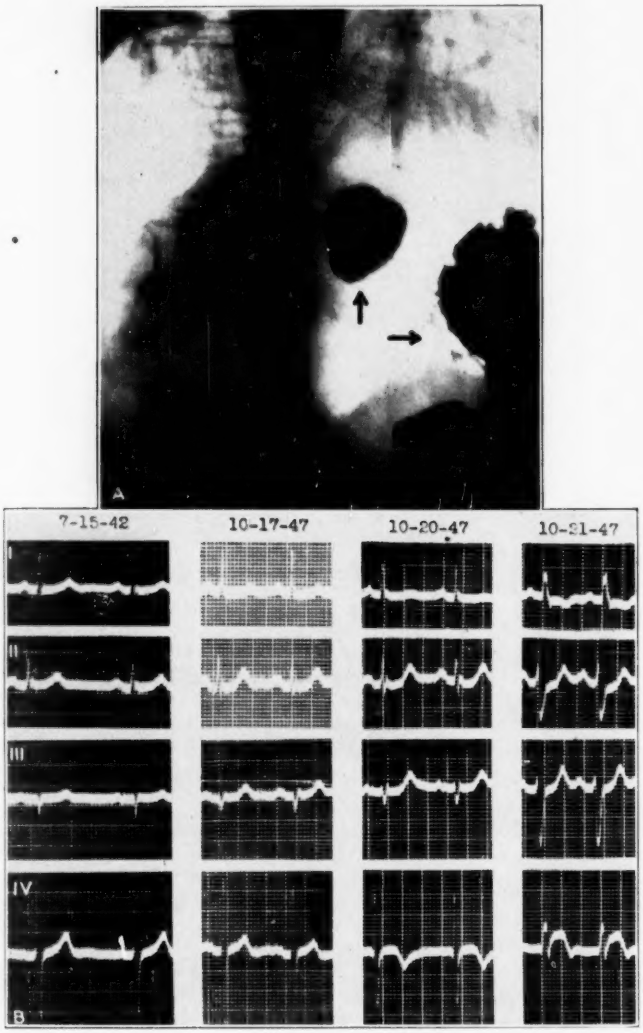

Fig. 3 (case 34).—*A*, moderate-sized hiatus hernia, demonstrated during premonitory phase of acute coronary occlusion. Indigestion and heartburn for two weeks; precordial and substernal pain for one week, relieved by standing. Acute coronary occlusion occurred day after roentgenologic examination of the stomach. *B*, electrocardiogram during premonitory phase (Oct. 17, 1947), normal and similar to old record of 1942. Subsequent tracings on October 20 and October 21 following an acute attack of thoracic pain show signs of infarction due to acute coronary occlusion.

and pressure radiating to the left shoulder and arm, lasting for several hours. Response to rest and the use of nitroglycerin was variable. Seven months before, after a severe attack of pain, the patient was hospitalized for six weeks and treated for acute coronary thrombosis. Weekly attacks of pain continued thereafter. Repeated electrocardiograms in the past were normal. During the past two years the patient had also experienced epigastric pain one hour after meals, relieved by milk and alkalis. A series of gastrointestinal roentgenograms had revealed a duodenal ulcer.

Physical examination of the cardiovascular system when the patient came under our observation was noncontributory except for moderately frequent ventricular premature beats. There was tenderness to deep epigastric pressure. Roentgenologic examination of the stomach revealed a hiatus hernia the size of a lemon (fig. 4*A*) and a markedly deformed duodenal bulb indicative of healed duodenal ulcer. The heart was normal in size and configuration, and the ventricular pulsations observed fluoroscopically were normal. The electrocardiogram, including multiple precordial leads, was entirely normal. No significant change occurred after the two step exercise test utilizing both the standard and the double number of trips (fig. 4*B*).

The absence of objective evidence of cardiac disease or coronary insufficiency in a man with a twenty year history of attacks simulating angina pectoris suggested that the hiatus hernia was responsible for the pain in the chest and left arm. It is likely that if coronary disease were present clinical evidence would become manifest after twenty years. Furthermore, the pain was not entirely typical of angina pectoris since it was unrelated to effort, was relieved by rest only after fifteen or twenty minutes, often lasted for several hours and responded to nitroglycerin infrequently. The normal two step exercise electrocardiogram ruled out the presence of organic coronary disease. The so-called "attack" of coronary thrombosis was in all probability an episode of pain from the hiatus hernia.

COMMENT

It is evident from our findings that uncomplicated hiatus hernia with few exceptions does not produce typical anginal pain on effort unless there is underlying coronary artery disease. If a patient with a known hiatus hernia experiences angina on effort, coronary artery disease should be suspected and will usually be confirmed by the two step exercise test.

We believe that in the past typical angina pectoris on effort has been attributed to a hiatus hernia too often because objective functional tests for coronary insufficiency were not carried out systematically. It is well known that in typical angina pectoris due to coronary artery sclerosis the physical and roentgenologic examination of the heart and the electrocardiogram may reveal no abnormalities in approximately one fourth of the cases. In order to confirm the diagnosis of coronary disease suggested by the symptoms, one must resort to the exercise or the anoxemia test. These tests will demonstrate the presence of coronary insufficiency when the thoracic pain is due to organic coronary disease. Our study further emphasizes the importance of utilizing these functional tests in patients with hiatus hernia who have referred thoracic pain.

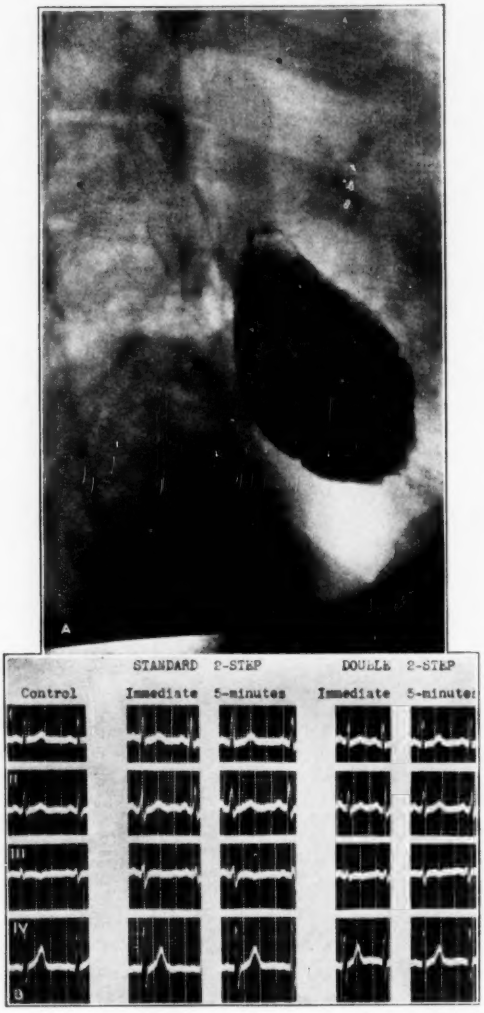

Fig. 4 (case 17).—*A*, hiatus hernia simulating angina pectoris and coronary occlusion. Recurrent substernal pain radiating to the left shoulder and arm for twenty years. *B*, electrocardiograms at rest and after standard and double two step exercise tests normal. The absence of objective signs of coronary insufficiency suggests that the pain is referred reflexly from the hernia to the chest.

We are of the opinion, after an extensive experience which has included thousands of tests with the two step exercise test and hundreds of trials with the 10 per cent oxygen test, that the two step test is much easier to perform and is safer. We have found no instance in which the 10 per cent oxygen test contributed additional information provided the double two step test was done when the standard two step test gave a negative result. Moreover, an occasional patient cannot complete the 10 per cent oxygen test but goes into collapse. If thoracic pain appears at all in the two step test it is likelier to be more severe in the anoxemia test. Finally, in some cases of coronary disease the reaction to the two step test will be positive when the reaction to the anoxemia test is negative.

There is no doubt that hiatus hernia can be associated with pain referred to the substernal and precordial region. As a rule, this pain is related not to effort but to meals and is not relieved promptly by rest or the use of nitroglycerin. It nearly always is accompanied with epigastric distress and indigestion. In only 6 of our cases in which the pain in the chest suggested a cardiac origin were we unable to elicit the objective signs of coronary disease. We concluded that in these patients the pain which simulated angina pectoris could have been produced solely by the hiatus hernia. In 3 of these patients, however, the cardiac work-up was not as complete as we desired.

Although we do not believe that hiatus hernia produces any disturbance in coronary circulation in the presence of normal coronary arteries, it is possible that it may act as a trigger mechanism in precipitating angina pectoris when the coronary arteries are sclerotic. A similar trigger mechanism has been demonstrated in the case of gallbladder disease. Stimuli through the upper abdominal visceral nerve pathways to the upper thoracic ganglions and spinal cord may be transmitted reflexly to the coronary arteries. The summation of such visceral stimuli and slight stimuli from the diseased coronary arteries may bring painful sensations in the thoracic wall to the conscious level. In the presence of normal coronary vessels, however, this mechanism must be rare unless another factor is present conducive to the development of coronary insufficiency, such as hemorrhage, anemia or shock. It is of interest that these factors of anemia, hemorrhage, shock, severe diarrhea and vomiting, which may occur in conjunction with hiatus hernia, are common precipitating factors of acute coronary insufficiency, particularly if coronary sclerosis is already present. Coexistence of the two conditions can be clearly verified in these cases, also, by the objective tests already enumerated.

SUMMARY

The differential diagnosis of hiatus hernia and coronary artery disease is of great practical importance, since hiatus hernia may produce symptoms simulating angina pectoris and occasionally acute coronary occlusion. The two conditions, which occur with increasing frequency in the same age group, may coexist; therefore, the finding of one does not exclude the other.

An intensive study of the cardiac status was made in 57 consecutive patients with proved hiatus hernia of the stomach. The examination included a complete history and physical examination, teleoroentgenogram, roentgenoscopy, resting electrocardiogram including augmented unipolar limb and Wilson precordial leads, the two step exercise test and the 10 per cent anoxemia test.

Evidence of organic heart disease was found in almost two thirds of the cases. On the basis of symptoms the patients were divided into four groups: group I, 15 patients with purely gastrointestinal symptoms; group II, 29 patients with both gastrointestinal and cardiac complaints; group III, 3 patients with only cardiac complaints, and group IV, 10 patients with symptoms attributable to anemia secondary to hemorrhage.

Four patients of group I had pain referred to the left side of the chest, but no evidence of coronary disease was obtainable. In 23 of the 29 patients in group II cardiac disease was demonstrated by the objective examinations. In the 6 remaining patients with thoracic pain suggestive or characteristic of angina pectoris organic coronary disease could not be demonstrated, which suggested that the hiatus hernia may have been the only factor initiating the pain in the chest through reflex pathways. These negative findings in the latter cases also indicate that the history alone is not a safe means of differentiating hiatus hernia from angina pectoris due to coronary disease. Nevertheless, the history may be helpful, for the occurrence of epigastric or substernal discomfort in the recumbent position which is relieved on standing is characteristic of hiatus hernia, whereas constant relation to effort and prompt relief by the use of nitroglycerin is more suggestive of coronary artery disease.

Hiatus hernia not infrequently may precipitate coronary insufficiency even in the presence of normal coronary arteries if hemorrhage, severe anemia or shock develops.

CONCLUSIONS

1. Hiatus hernia and coronary artery disease can be distinguished from each other, and when they coexist the clinical importance of each can be properly evaluated.

2. This study emphasizes the value of objective functional tests for coronary insufficiency, especially the two step exercise test, in the dif-

ferentiation of these two conditions. A positive reaction to the two step test may be the only objective sign of coronary artery disease.

3. Although uncomplicated hiatus hernia does not produce typical anginal pain on effort unless there is associated coronary artery disease, exceptions do occur and objective functional tests are necessary for evaluation of the thoracic pain.

4. Hiatus hernia may produce pain referred through reflex autonomic pathways to the wall of the chest and the left arm. The functional tests in such instances do not yield evidence of coronary insufficiency.

ABSTRACT OF DISCUSSION

DR. RALPH C. BROWN, Chicago: These papers are timely. While a rather voluminous literature has appeared throughout the years, there is a lack of general awareness of the frequency of these hernias as well as of the important role they frequently play in diagnosis. My impression is that until recent years clinicians were inclined to disregard the small hernias so frequently seen in the course of roentgen examinations in which the Trendelenburg position and right and left turning of the patient, with simultaneous deep inspiration, were routine procedures. Our interest was concentrated more particularly on the large hernias causing dysphagia or hemorrhage or on the distress in the lower thoracic area associated with changes in posture.

However, between 1938 and 1943 a series of illuminating papers appeared, especially those of Moersch, Stuart, Harrington, Huber and Andresen and Chester Jones, and our diagnostic horizons were greatly enlarged. Harrington's survey showed that in 295 cases of diaphragmatic hernia in which he had operated there had previously been made an average of three erroneous diagnoses. In order of frequency these were cholecystitis, cholelithiasis, gastric ulcer, duodenal ulcer, hyperchlorhydria and anemia. However, we are still in need of facts from clinical, pathologic and roentegenologic sources. I think the majority of hiatus hernias are symptomless. Why do hernias of the same size and precisely the same appearance behavé so differently?

Dr. Masters and his associates have made a detailed and exacting differential diagnostic study of coronary artery disease and hiatus hernias, and their conclusion that uncomplicated diaphragmatic hernia gives no objective evidence of coronary artery disease is a valuable observation. It seems probable that careful history taking, especially in regard to the conditions under which substernal pain arises, the area of radiation of pain, the effect of posture and, especially, of nitroglycerin, as well as electrocardiographic studies will limit to a relatively small group of cases those in which serious difficulty in differential diagnosis will arise. In Dr. Brick's series of 308 patients, 28 per cent had substernal pain or distress, whereas only 6 per cent had pain in the arm or hand; there is no information on how many in this 6 per cent group had radiation of pain to the flexor surface of the arm. In Chester Jones's 128 cases substernal pain occurred in 39 patients and pain in the arm or hand in only 12. The frequent association of coronary artery disease and hiatus hernia reported by Dr. Richards and Dr. Crockett emphasizes the importance of the technic of differential diagnosis described by Dr. Masters and his associates.

The data presented in Dr. Brick's statistical study are of great interest, especially those dealing with gastrointestinal lesions associated with hiatus hernia and with the fact that hiatus hernia was the second most common diagnosis made,

duodenal ulcer being first. I should like to ask Dr. Brick if he has any notes on the incidence of hemorrhage in his series and how many gastric ulcers were found in the herniated part of the stomach. Cases of hiatus hernia associated with either massive hemorrhage or slow, exsanguinating hemorrhage may present a difficult clinical problem, primarily because, in my experience, it is so difficult to visualize by roentgen ray the peptic ulcer which one suspects exists, probably at the point of greatest constriction, as the probable source of the hemorrhage.

Dr. Manfred Kraemer, Newark, N. J.: My interest in diaphragmatic hernia began in 1933 when I saw a woman 54 years old who had a large hernia of the stomach on the left side and an equally large one of the colon through the right side of the diaphragm. These hernias had been discovered by Dr. Ernst May in 1927 and were reported by him in a radiologic journal. This was the first reported bilateral diaphragmatic hernia diagnosed by roentgen ray. Repair had been advised in 1927 and was again urged in 1933. She refused operation both times. She was an insurance broker and worked every day despite some substernal pressure. She was still able to work when I last heard of her in 1942. She was then 65 years old. So far as I know, she is still alive and carrying a good part of her colon and stomach in her chest. This case emphasizes that surgical treatment of diaphragmatic hernia need be resorted to only occasionally.

One most careful roentgenologic examination does not exclude the presence of a diaphragmatic hernia. In every case in which hernia is suspected and especially in unexplained cases of hemorrhage of the upper part of the intestinal tract we repeat our roentgen examination for hernia if none was found at the first examination.

The best position for demonstrating a diaphragmatic hernia is the prone Trendelenburg position, with the stomach full of barium sulfate suspension. We recently used both the prone Trendelenburg and the supine positions in a series of 600 consecutive cases. The prone position was the most accurate. In every case in which the hernia was demonstrated in the supine position it was also seen on the roentgenograms made in the prone position, but in several cases in which it was shown in the prone position it was missed in the supine position. Diaphragmatic hernia is often missed because a roentgenogram of the patient in the prone Trendelenburg position with a full stomach is not taken routinely in every gastrointestinal examination. Although there is a general impression that these hernias can be operated on only transthoracically, Dr. Edward Sprague has successfully repaired a number of them transabdominally. As there is often disease elsewhere in the abdomen, coexistent with diaphragmatic hernia, it is safer to use the abdominal approach and to explore the abdominal cavity before repairing the rupture.

Dr. Donovan C. Browne, New Orleans: I wish only to stress one point, that these patients bleed and that this hemorrhage or bleeding may be exsanguinating. We have now had 6 of them in the gastroscopic clinic, in 4 of whom there was actually exsanguinating hemorrhage. Two of these were simply anemic and continued so. Among the 6 we have been able to demonstrate gastroscopically the bleeding points, and those doing the gastroscopy appreciate the difficulty and the rarity of this demonstration. The patients which we have had have all been beyond 60 years of age, and all have been obese.

Dr. Irving B. Brick, Washington, D. C.: In answer to Dr. Browne's question, we have 5 patients among our 308 who showed an ulcer in situ or an ulcer of the hiatus hernia. As to incidence of hemorrhage, I am not able to give the figures in this series. However, in another similar study, Dr. Hampton and Dr. Sahler, at the Massachusetts General Hospital, reported that in 223 cases they found evidence of bleeding in 8 per cent. As to the question of asymptomaticity of these lesions, there is no doubt that hiatus hernias, either small or large, may occur in patients

who have no symptoms whatever and the presence of the hernia is a routine coincidental finding. However, there is not in the available literature a study of a sizable group of patients without gastrointestinal symptoms in the 50 to 70 age group in which specific roentgenologic examination for hiatus hernia was undertaken. Such a report is required so that some idea can be obtained to contrast with the incidence of hiatus hernia in patients who are having such symptoms that gastrointestinal examination is indicated. Such a study is certainly required so that we can get some idea of whether or not in the asymptomatic group of similar age the percentage of hiatus hernias is as high as in the symptomatic group. It is undoubtedly not as high, but there will probably be found a sizable percentage.

Dr. Arthur M. Master, New York: Dr. Browne said that the history is of great value. It is probably the most valuable single differential point between hiatus hernia and coronary disease; yet I believe that the objective symptoms must be used frequently and sometimes one must depend on them solely. A very important differential sign is position of the body. In a patient with hiatus hernia the pain is worse on lying down or bending over and is relieved on standing up; but we advise patients with severe coronary disease not to bend over to tie their shoe laces since this frequently brings on their pain! I can go further and tell you the story of a man of 63 years of age, suffering from severe coronary disease, whose sternal pressure was induced by bending forward, whereas standing up and stretching relieved the pain. He died of a heart attack and was given postmortem examination. Severe coronary disease was found but no hiatus hernia. Similarly, patients with hiatus hernia may appear to be suffering from sclerosis of the coronary arteries. In the 57 cases we reported, many patients gave a distinct history of an anginal syndrome due to effort and yet they did not have coronary disease. They complained of pressure in the sternum while walking or climbing stairs, and yet we have shown that the only lesion from which they suffered was the hiatus hernia. A long follow-up has substantiated this. Hence, in summary, I repeat that although the history may be the most important single differential sign objective tests such as we have utilized are often necessary.

GRANULAR CELL MYOBLASTOMA OF THE ANTERIOR RECTUS SHEATH

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GRANULAR cell myoblastoma and rhabdomyoma are the two tumor types, both rare, arising from striated muscle cells. The myoblastomas were described originally as "Myoblastenmyome" by Abrikossoff [1] in 1926. At that time he regarded them as neoplasms due to faulty excessive regeneration subsequent to injury of striated muscle. In 1931, realizing that some of these tumors arose in areas devoid of striated muscle, he postulated [2] their origin from primitive myoblasts representing embryonal rests. He classified myoblastomas into four histopathologic types: (1) those consisting of granular myoblastic cells without the presence of striation, (2) the same type with the addition of occasional faint striations, (3) hypertrophic forms consisting of large multinucleated cells and (4) malignant myoblastic myomas consisting of pleomorphic cells, some areas showing differentiation and striations and others frankly sarcomatous in appearance.

On gross examination, myoblastomas are tumors varying in diameter from 0.5 to 2 cm., although they occasionally may be up to 10 cm. Some are well circumscribed or encapsulated, and others are poorly demarcated and may infiltrate into neighboring tissues.[3] They are usually round but may be nodular or lobulated. The cut surface is firm, appears relatively avascular, is yellowish gray to pearly white and may present a finely lobulated appearance.

They consist, on microscopic examination, of large polygonal cells with deep-staining, vesicular nuclei, large amounts of granular and acidophilic cytoplasm and occasional protoplasmic processes of fibrils. Some of these cells may be rounded and others spindle or cylindric shaped. Mitosis is usually absent. On low magnification the cells appear to occur in groups, but under high power they lie in ribbon-like syncytial masses, with thin strands of connective tissue interspersed

From the Surgical Service, Hospital for Joint Diseases, New York.

1. Abrikossoff, A.: Ueber Myome, ausgehend von der quergestreiften willkürlichen Muskulature, Virchows Arch. f. path. Anat. **260**:215, 1926.

2. Abrikossoff, A.: Weitere Untersuchungen ueber Myoblastenmyome, Virchows Arch. f. path. Anat. **280**:723, 1931.

3. Crane, A. R., and Tremblay, R. G.: Myoblastoma, Am. J. Path. **21**:357 (March) 1945.

among many of them. These fibers in the stroma are characteristic, since they may be traced, on careful study, to the myoblasts.[4] Cross or longitudinal striations are seen infrequently and, when apparent, according to Klemperer,[5] are formed by a transverse arrangement of the granules at the periphery of the bands. Actually there is a strict separation of the muscle fibers and tumor cells. Occasionally granular cell myoblastomas are found in which the cells have large nuclei and are arranged in lobules separated by capillaries, giving the tumor the appearance of an endocrine gland, the so-called organoid type described by Horn and Stout[6] and by Hartz.[7] According to Stout,[8] the only feature which distinguishes the malignant myoblastoma from the benign is a larger nucleus with a larger nucleolus. Even this feature may be lacking in the primary tumor and may appear only in the area of metastasis. Apparently there are no reliable characteristics of malignancy which are always present in the primary tumors. Howe and Warren[9] described atypism of the cells, excessive mitotic figures, spindle-shaped cells and local invasion as signs of malignant myoblastomas.

Granular cell myoblastoma is differentiated from xanthoma by the fact that there is no fat in the former and from rhabdomyoma and rhabdomyosarcoma, which are composed of adult striated muscle elements.

Although myoblastoma is regarded as an essentially benign tumor, many cases of malignant myoblastomas have been reported. Geschickter[4] described a case of myoblastoma in which metastasis developed five years after excision of the primary lesion. Howe and Warren[9] in 1944 described 10 new cases of which 5 showed malignant properties, the tumor metastasizing to the lungs in 3 of the latter, and reviewed over 100 cases of which 11 per cent showed malignant characteristics. In 1947 Stout[10] described two myoblastomatous tumors of the soft parts which metastasized and subsequently caused

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4. Geschickter, C. F.: Tumors of Muscle, Am. J. Cancer **22**:378 (Oct.) 1934.

5. Klemperer, P.: Myoblastoma of the Striated Muscle, Am. J. Cancer **20**:324 (Feb.) 1934.

6. Horn, R. C., Jr., and Stout, A. P.: Granular Cell Myoblastoma, Surg., Gynec. & Obst. **76**:315 (March) 1943.

7. Hartz, P. H.: So-Called Granular Cell Myoblastoma of the Thigh With Organoid Structure, Am. J. Clin. Path. **14**:582 (Nov.) 1944.

8. Stout, A. P.: Granular Cell Myoblastoma of the Lumbar Region, Texas State J. Med. **41**:580 (March) 1946.

9. Howe, C. W., and Warren, S.: Myoblastoma, Surgery **16**:319 (Sept.) 1944.

10. Stout, A. P.: Sarcomas of the Soft Parts, J. Missouri M. A. **44**:329 (May) 1947.

death. In the same year Powell[11] reported 2 cases; in 1 the tumor metastasized from a subcutaneous nodule involving the ovaries and retroperitoneal tissues, and the other resulted fatally because of local invasion and erosion of a large blood vessel.

Myoblastomas may be found in almost any part of the body. In most of the cases reported and summarized in the literature the tumor was located in the tongue, skin, subcutaneous tissues including the breast,[12] muscles of the extremities, maxilla, lip, trachea or bronchi, ear, alveolar process and anus. Single cases have been reported in which it occurred in the bladder,[13] esophagus, lacrimal sac, floor of the mouth, pharynx, spermatic cord, urethra, vulva, thoracic wall[14] and the aponeurosis of the external oblique muscle.[15] Those located under mucous membranes or skin have a decided tendency to be associated with hyperplasia and epidermoid carcinoma of the overlying epithelium.[9]

The following case is the first one reported in which the tumor occurred in the sheath of the rectus abdominis muscle. In this location it resembled clinically the more common desmoid tumor.

REPORT OF A CASE

In March 1947 a white man, 57 years of age, of Polish descent and a butcher by occupation, first noticed a small, hard, nontender lump in the anterior abdominal wall. He gave no preceding history of trauma. His family history and past history were noncontributory. The lump gradually increased in size. The patient entered the hospital in September 1947, and a wide excision of a scirrhous-like, fairly well demarcated tumor, about 2 cm. in diameter, located about 5 cm. below and 3 cm. to the left of the umbilicus and firmly attached to the anterior rectus sheath, was performed. The tumor, well covered with fat, was excised along with a generous margin of the anterior rectus sheath, the underlying segment of rectus muscle and local preperitoneal fat. Closure of the fascia was effected with alternating interrupted no. 2 chromic surgical gut and no. 30 stainless steel wire sutures. The postoperative course was uneventful, and the wound healed by primary union.

Pathologic Report.—Grossly, the specimen consisted of an irregular mass of fatty tissue from the panniculus adiposus, together with a strip of fascia of the rectus muscle on the under surface of the specimen and two fragments of muscle tissue from the rectus, deep to the tumor. Within the fatty tissue in one area there was a localized, but not well circumscribed, scirrhous tumor node. The latter measured approximately 2 cm. in its greatest diameter. The tumor sat on the

11. Powell, E. B.: Granular Cell Myoblastoma, Arch. Path. **42**:517 (Nov) 1946.

12. Simon, M. A.: Granular Cell Myoblastoma, Am. J. Clin. Path. **17**:302 (April) 1947.

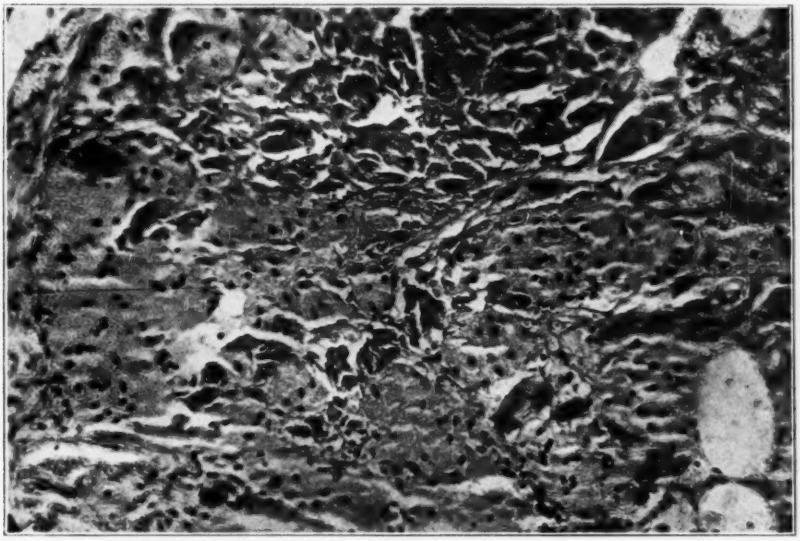
13. Ravich, A.; Stout, A. P., and Ravich, R. A.: Malignant Granular Cell Myoblastoma Involving the Urinary Bladder, Ann. Surg. **121**:361 (March) 1945.

14. Orr, T. G., Jr.: Myoblastoma: Case, Ann. Surg. **122**:122 (July) 1945.

15. Grayzel, D. M., and Friedman, H. H.: Myoblastoma of the Thoracic Wall, Arch. Path. **31**:512 (April) 1941.

fascial sheath and appeared to be infiltrating it locally, but there was no obvious gross evidence of penetration beneath it, nor was there gross evidence of extension of the tumor into the muscle fragments. The tumor tissue was mottled yellow gray in appearance and, as noted, was extremely hard.

Microscopically, the sections showed the tumor to be composed of lobules and strands of closely approximated cells with granular cytoplasm. The granular appearance of the cytoplasm did not reflect the presence of fat. The tumor lobules in places were separated by bands of collagenous connective tissue. At the periphery of the tumor the strands of connective tissue were invading the fat locally. The sections of the muscle tissue showed no evidence of extension of the tumor.



Photomicrograph showing tumor cells with small, deep-staining nuclei and granular cytoplasm. Hematoxylin and eosin; × 125.

The tumor in question resembled a myoblastoma, even though it developed externally to the rectus sheath. The pathologic diagnosis was myoblastoma of the anterior abdominal wall.

COMMENT

In the case described the tumor may be classed as corresponding to the type I of Abrikossoff.[1] Its extramuscular location suggests its origin from an embryonal rest.

Some myoblastomas are true neoplasms, while others are considered as originating from a local degeneration of skeletal muscle. Geschickter [4] expressed the opinion that the myoblastomas represent a phase in the histogenesis of voluntary muscle. He described the transition of this muscle from a syncytial stage in the mesenchyme to a myoblastic

stage and thence to a myofibrillar stage in which the developing fibers show first beading and then cross striations. In this last stage multinucleated cells are common. Stout [8] stated the belief that some represent a development from undifferentiated mesenchymal cells of an abortive form of striated muscle cells. Such granular cells normally do not exist.

These tumors show no relationship to sex and may occur at all ages. However, they are most common in the third to fifth decade. Klemperer [5] stated that they may occur at birth inasmuch as the so-called congenital epulis of the newborn is histologically a myoblastoma. He also stated that the male sex is more frequently affected, the ratio being about 2 to 1.

The treatment of choice is surgical extirpation. Recurrence may follow incomplete removal. These tumors are markedly radioresistant. Horn and Stout [6] described a case in which a myoblastoma of the tongue survived a sufficient dose of radium to produce necrosis of the tongue. However, in general, the outlook is favorable. The great majority of granular cell myoblastomas are benign and do not recur after excision.

SUMMARY

1. Granular cell myoblastoma is a rare tumor arising from striated muscle cells.

2. It is regarded as an essentially benign tumor; however, malignant forms with local infiltration and distant metastasis have been described.

3. This tumor may occur in any part of the body, and when it is found in areas devoid of striated muscle its origin is thought to be from embryonal rests.

4. The treatment is surgical excision. The tumor is radioresistant.

5. A case of granular cell myoblastoma of the anterior rectus sheath, apparently arising from an embryonal rest, is described.

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TREATMENT OF PEPTIC ULCERATION BY VASCULAR LIGATION

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THERE appears to be little doubt that the fundamental factor in the production of peptic ulcer is the gastric juice, and there is an increasing body of evidence which suggests that it is the hydrochloric acid of the gastric secretion which is the element concerned. The gastric juice does not normally digest the stomach because it is buffered by the food and also by swallowed saliva, the secretion of the alkaline glands of the antrum and cardia and the regurgitation of duodenal secretion. The danger period occurs when the stomach is empty of food and especially during the hours of sleep. It is probable that patients who suffer from gastric or duodenal ulcer are the subjects of a derangement of gastric secretion, probably resulting from a functional nervous cause, as a result of which secretion of gastric juice is more profuse during the interdigestive periods than in the normal subject. In the normal person secretion during the night hours is scanty and the small amount of gastric juice is neutralized by saliva, duodenal secretion and alkaline antral secretion; in the person with ulcer the quantity of gastric juice secreted may be such that this neutralization is not possible and the mucosa is exposed to the action of pure gastric juice or of only partly diluted gastric juice. The brilliant American experimental work, especially that of Dragstedt, has shown that undiluted gastric juice will digest practically any kind of living tissue, and the histamine-wax experiments carried out in the University of Minnesota have demonstrated that if the secretion of gastric juice is kept up continuously chronic ulceration of the stomach and duodenum can be readily produced.

If these views are correct, it is natural that in the search for a cure for peptic ulceration the main object should be the cutting down of the quantity or of the acidity of the gastric juice. It is probable that in the not far distant future the physician will discover some means of doing this effectively, but in the meantime a certain proportion of cases resist his treatment and surgical measures are required. I shall not recapitulate the various phases through which the surgical treat-

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ment of peptic ulceration has passed. In this country twenty-five years ago gastroenterostomy held the field; its place now has been taken by partial gastrectomy. The clinical results of gastroenterostomy were satisfactory in many cases, especially in those in which an organic stenosis was present. The failure of this operation has been due to the frequency with which it has been followed by ulceration of the jejunum, especially in young subjects with a patent pylorus and high gastric acidity. British surgeons were much slower in adopting gastrectomy in the treatment of peptic ulceration than those of America and the Continent, and many, I know, still have qualms about performing it in cases of duodenal ulcer in which the operation appears unnecessarily drastic for a lesion which is small in size and not particularly lethal. There can be no doubt, however, that the results of the operation are satisfactory in a large proportion of cases. It produces its effects mainly by removing a large part of the acid-secreting portion of the stomach, by removing the mucosa of the antrum, which is probably the source of a hormone concerned in gastric secretion, and by removing the site of ulceration. No doubt, the more rapid emptying of the stomach and the free regurgitation of intestinal juices are also beneficial.

I have said that qualms are still felt by some surgeons about the routine performance of this operation in the treatment of duodenal ulcer. These qualms are occasioned by the fact that the operation is a severe one and is bound to be attended by a certain mortality, which may be taken as being between 5 and 10 per cent. Secondary ulceration has not been completely eliminated, and secondary ulceration after gastrectomy is a much more formidable problem than that after gastroenterostomy. In addition, fears have been expressed about the effects of this operation on the patient's nutrition and vitality. How far these fears are justified does not seem to be definitely settled; the patients do not usually put on much weight after the operation, but the general health, as a rule, remains satisfactory. However, occasional cases of persistent anemia are met with, and attention has been directed recently to the incidence of hypoglycemia after meals, sometimes leading to loss of consciousness, this condition being due to dumping of the food in the upper coils of the small intestine because of the rapid evacuation of the stomach. As a result of these imperfections, the search for a better form of operative treatment still continues, and this has manifested itself from time to time by the introduction of such procedures as the various operations at the pylorus, the Connell fundusectomy, the Devine method of gastric exclusion and, recently, particularly by the revival of interest in vagotomy. Attempts have also been made to reduce the quantity and acidity of the gastric secretion by attacks on the blood vessels of the stomach. This procedure has only aroused interest in recent years and has

not yet been extensively tried out. The pioneer in this field was Hey,[1] of Manchester, but his only article on the subject was published in an American journal which I have only recently been able to obtain. Much work has been done by Somervell[2] on somewhat similar lines, and it is his method which I have employed in the cases on which I have to report.

Somervell first distinguished himself by climbing with a companion nearly to the top of Mount Everest and reaching a greater height without the help of oxygen than anyone has ever done. He then became a medical missionary in the south of India. In the region in which he worked duodenal ulcer was a common malady. He attributed this to the nature of the diet, which consisted mainly of rice and curry or tapioca and curry. Dietetic treatment outside the hospital was not possible owing to the extreme poverty of the native population, and the great majority of the patients with peptic ulcer were treated surgically. In 1936 he and a colleague reported 2,500 operations for gastric, duodenal and jejunal ulcers. He then performed gastroenterostomy as the routine operation for duodenal ulcer if there was stenosis or a normal or low acidity. In cases with a high acidity and no stenosis he performed a gastrectomy with pyloric exclusion after the Finsterer method, leaving the pyloric antrum and its mucosa. The mortality in gastrectomy for 81 cases of duodenal ulcer was 1.3 per cent. He has now performed over 2,000 operations for duodenal ulcer. Writing in 1942, he stated that he had been led to modify his operative procedure during the preceding five years. Instead of performing a partial gastrectomy in the cases with a high acidity he had adopted the procedure which he called a "physiological gastrectomy." In all cases with an acidity of the gastric juice of over 60 he treated the condition by ligation of a large proportion of the blood vessels of the stomach with the addition of a gastroenterostomy. In those with an acidity near the normal he performed a gastroenterostomy alone. It is interesting to note that he found the operation of gastroenterostomy a reliable method of reducing the gastric acidity in his patients. He found that the operation did not reduce the acidity immediately but did so effectively in the long run. His reason for adding the ligation of the gastric blood vessels to the gastroenterostomy was the fear of the development of stomal ulceration during the interval, possibly a year or eighteen months, which it took the gastroenterostomy to produce its effects. He found that if the gastric blood vessels were ligated as well the acidity dropped immediately and this obviated the risk of jejunal ulceration. He treated a few patients

1. Hey, W. H.: Proc. Interst. Postgrad. M. A. North America, 1937, p. 291.
2. Somervell, T. H., and Orr, I. M.: Brit. J. Surg. **24**:227, 1936. Somervell, T. H.: ibid. **30**:113, 1942; **33**:146, 1945.

by ligature of the blood vessels alone and obtained a satisfactory fall in the acidity in most of these, but he considered gastroenterostomy to be an essential part of the operative treatment. Among over 380 patients operated on by himself and his colleagues there was only one death. The operation consists in tying four of five or five of six of the blood vessels along the greater curvature from near the pylorus to the point where the left gastroepiploic artery reaches the stomach. This is done first on the anterior and then on the posterior surface after making a window in the mesocolon. On the lesser curvature all the vessels are tied except for one small vessel near each end of the curvature; this also is done on both anterior and posterior surfaces. He found that the resulting fall in acidity persists for at least five or six years. He suggested that this fall is due to the loss of blood supply, which causes the parietal cells to secrete less acid or possibly many of these cells to die or perhaps causes the hydrochloric acid to be secreted in a more dilute form. In other organs, when a proportion of the blood vessels are tied or destroyed, collateral channels enlarge and the blood supply is restored. Somervell suggested that this does not take place in the case of the stomach because the blood supply is already overadequate and after four fifths have been tied the remainder is sufficient for the stomach's needs without the necessity for dilation.

This seemed to me a method of treatment which ought to be tried out, and I performed the operation in 47 cases of duodenal ulcer and then stopped to await results. Most of the patients came from the medical side of the Royal Infirmary, and the majority had undergone a prolonged course of medical treatment. In all cases the vessels were tied as in Somervell's method and a gastroenterostomy performed. In cases with an acidity near the normal Somervell performs gastroenterostomy alone, but it was thought best to tie vessels in all cases as it seemed reasonable to believe that the ulcer would benefit from a fall in the acidity even when the acidity was within the normal limits before the operation. A fractional test meal was carried out before the operation and again at an interval of eight to twenty-one days after operation. The second test meal usually showed a definite fall in the acidity of the gastric juice, with complete achlorhydria in a few cases. At the end of 1946 a follow-up of 29 patients who had been operated on between December 1942 and June 1945 was carried out, that is, after an interval of from one and a half to four years. Twenty-four patients reported for examination, and 5 more replied to a questionnaire. Of the 24 reporting, 17 consented to a further test meal; 10 of these 17 had suffered from pyloric stenosis before operation, while in 7 there was no stenosis.

At the time of the investigation, November and December 1946, 27 of the 29 patients were back at full work. Twenty-seven reported themselves as free from gastric symptoms, but on more thorough questioning 2 admitted occasional heartburn and 4 said they occasionally vomited after a meal. Two were not working, 1 on account of a severe hematemesis two years after operation and 1 for some complaint unrelated to his gastric condition. Nearly all the patients had gained considerably in weight; 1 had gained as much as 56 pounds (25.4 Kg.), and 2 had gained 42 pounds (19 Kg.). The average gain in weight was 19 pounds (8.6 Kg.), and there was no great difference between those who had and those who did not have pyloric stenosis at the time of operation.

My first impression from this investigation was that the results of the operation were reasonably satisfactory, but further experience and the results of the late test meal have altered this opinion. In 2 of the patients in this group of 29 stomal ulceration has since developed, and they have been subjected to partial gastrectomy. In addition, a third patient, not included in the group, was operated on for the same condition, so that in this small series within four years from the time of operation there have already been 3 cases of jejunal ulceration. In the most recent of these cases and in a further case to be referred to presently special note was made of the appearance of the gastric blood vessels at the second operation. There was a definite dimunition in the number of the vessels on the greater curvature, but on the lesser curvature fresh vessels had opened up and had taken the place of those which had been ligatured, so that the appearance of the vessels in this region did not differ appreciably from the normal. A recent patient was of special interest. He had been operated on by a colleague in 1932 for symptoms of duodenal ulcer. The record of this operation could not be traced, but, as was found later, it was either a mere exploration or a minor procedure at the pylorus. His symptoms soon recurred and were associated with attacks of hematemesis. He was treated in the medical wards on several occasions and was admitted there in February 1946 after a severe hematemesis. He then had high acidity of the gastric juice. The roentgenologic examination showed only slight deformity of the duodenal cap. I saw him then and advised further operative treatment. At operation I was unable to demonstate a definite ulcer and I treated the patient by ligature of the gastric vessels alone, without gastroenterostomy. The test meal shortly after operation showed a considerable fall in the acidity. He remained well for six months, when his symptoms returned and he had three further attacks of bleeding. On investigation, about fifteen months after ligature of the gastric blood vessels, the acidity of the gastric juice was found to have risen almost to the preoperative level. At

a further operation on June 17, 1947, a cicatricial mass was found in the first part of the duodenum and partial gastrectomy was performed. This was an instructive case because it showed that the vascular ligation alone did not bring about a permanent reduction in the gastric acidity and did not protect the patient from further ulceration. The appearance of the gastric blood vessels was similar to that in the previous case; there was a diminution in the number of vessels on the greater curvature, but the vascularity of the lesser curvature did not differ appreciably from the normal owing to enlargement of collateral blood vessels. On section of the wall of the stomach, the vessels bled profusely and I had rather more trouble than usual in ensuring complete hemostasis at the suture line.

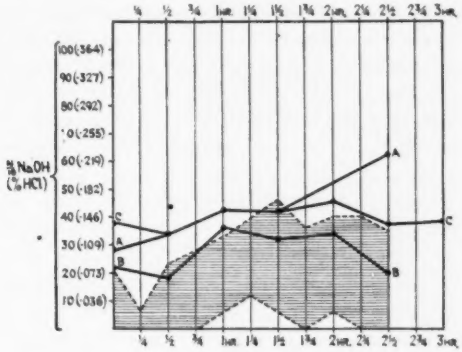
As regards the late effect of vascular ligation on the gastric acidity, 17 patients, as already mentioned, submitted to a further test meal which was carried out between one and a half and four years after operation. This showed that the acidity of the gastric juice had now returned almost exactly to the preoperative level. I was surprised that the gastroenterostomy by itself had not brought about a permanent reduction in the acidity. Somervell found in his Indian patients that this invariably took place, though it might be as long as a year or eighteen months before the full effect was produced. Heuer and Holman,[3] of Cornell University, however, in a careful study of 75 American patients who had had gastroenterostomy performed, mostly for duodenal ulcer, found that 92 per cent showed free hydrochloric acid equal to and, in the large majority, greater than normal, so that the operation of gastroenterostomy must produce its beneficial results in some other way than by reducing the acidity, possibly by permitting more ready emptying of the stomach.

This operation of vascular ligation in my hands has therefore been a failure. There have been three stomal ulcers in 47 cases within four years of operation, ligature of the gastric blood vessels by itself in 1 case was soon followed by further trouble from ulceration and examination of the gastric juice after one and a half to four years showed that it had risen practically to the preoperative level. Inspection of the stomach at a second operation after an interval showed that fresh vessels had developed on the lesser curvature. It seems to me that the effects of this method of vascular ligation are only temporary and that fresh anastomotic vessels develop in the wall of the stomach so that there is no permanent reduction in the vascularity of the stomach. The beneficial effects of the operation in European patients are probably entirely due to the gastroenterostomy. The

3. Heuer, G. J., and Holman, C.: Ann. Surg. **118**:551, 1943.

liability to stomal ulceration is no less than after gastroenterostmy alone.

It remains to reconcile these results with the good results that Somervell was able to obtain. I have not had the privilege of seeing him perform this operation, and there may have been some defect in my technic. However, I followed his description of the method to the best of my ability, and in a proportion of cases, at least, the bluish cyanotic appearance of the stomach showed that its blood supply had been considerably reduced. It seems to me to be more likely that the Indian patients and the Scottish patients suffered from different types of the disease. Owing to the impossibility of applying medical treatment, Somervell operated on all patients with a high acidity who had had symptoms for more than a year, whereas our cases were of a chronic



Constructed curves from personal cases in which treatment was by Howard Somervell's method. *A*, before operation; *B*, eight to twenty-one days after operation; *C*, follow-up.

type, and many of the patients had undergone prolonged courses of medical treatment. In the cases in India, where the population lives on a diet of rice and curry, the dietetic factor may play the main part in the production of the ulcer, while in European patients the dietetic factor is probably less important than some other factor, possibly neurogenic. It may be that in the conditions present in the stomach in the Indian cases a diminution of the blood supply has a more striking and lasting effect.

In conclusion, I wish to refer to the method of Hey, which differs in several respects from the operation of Somervell. The primary object of Hey's method is a sympathetic denervation of the stomach so that the unopposed vagi may bring about more rapid emptying. In order to accomplish this, all the main vessels of the stomach are tied, and the effect is to bring about a more complete devascularization than that

obtained by the method of Somervell. Hey originally divided the blood vessels, but as a result of the work of Lee of Baltimore he now employs a quadruple ligature of linen thread or silk which is a more effective barrier to sympathetic regeneration than simple division. His work is based on about 150 animal experiments. All the arteries on both curvatures are tied from the esophagus to within an inch (2.54 cm.) of the pylorus. The pylorus and its neighborhood are left untouched on account of four or five vagal branches descending in the lesser omentum to the pylorus. A gastroenterostomy is performed in addition because of the tendency to the development of stenosis after healing of the ulcer. Hey regards the effects of the devascularization as only temporary. He finds that the acidity of the gastric juice is reduced for an average period of nine months and thereafter tends to rise, though probably not to the preoperative level. In a personal communication he has very kindly supplied me with the details of his method and informs me that he intends to publish the results in his first 500 cases before long. I am sure that these will be of great interest, and I wish to make it clear that my criticisms of the operation of which I have had personal experience have no application to the method of Hey, which is fundamentally different in principle.

ASPIRATION OF BLOOD FROM PERICARDIUM IN TREATMENT OF ACUTE CARDIAC TAMPONADE AFTER INJURY

Further Experience, with Report of Cases

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FIVE years ago we [1] reported our experience with the aspiration of blood from the pericardial cavity in the treatment of acute cardiac tamponade due to hemopericardium. At the time of our report, the results led us to the conclusion that aspiration of the pericardial contents is the treatment of choice for hemopericardium and that open operation should be resorted to only if bleeding continues or is resumed. Blau,[2] Anderson and Starbuck [3] and others have reported successful results with nonoperative treatment.

Since our previous report, there has been an improvement in the mortality rate associated with the operative treatment of wounds of the heart. In 1944 Linder and Hodo [4] reported a mortality of 43 per cent in 28 cases, a rate which is almost identical with Elkin's mortality of 42 per cent in 38 cases, reported in 1941.[5] However, it is of interest that in the last 9 cases reported by Linder and Hodo there were no deaths, but that in 3 of them there was cardiac tamponade without any demonstrable wound of the heart itself. A subsequent report by Elkin [6] is even more encouraging in that there was a mortality rate of only 22 per cent in 23 cases of stab wounds of the heart treated by operation. Part of the improvement was attributed by these observers to vigorous

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1. Blalock, A., and Ravitch, M. M.: A Consideration of the Nonoperative Treatment of Cardiac Tamponade Resulting from Wounds of the Heart, Surgery **14**:157 (Aug.) 1943.

2. Blau, M. H.: Wounds of the Heart, Am. J. M. Sc. **210**:252, 1945.

3. Anderson, H. M., and Starbuck, R. W.: Conservative Therapy of Cardiac Tamponade Resulting from Wounds of the Heart, J. Florida M. A. **33**:86, 1946.

4. Linder, H., and Hodo, H.: Stab Wounds of the Heart and Pericardium, South. M. J. **37**:261, 1944.

5. Elkin, D. C.: Diagnosis and Treatment of Cardiac Trauma, Ann. Surg. **114**:169, 1941.

6. Elkin, D. C.: Wounds of the Heart, Ann. Surg. **120**:187 (Dec.) 1944.

fluid replacement, the experimental basis for which was presented by Cooper, Stead and Warren.[7]

Since our last report, we have admitted to the hospital a total of 8 patients with cardiac tamponade due to wounds of the heart. Seven of these patients were treated by pericardial aspiration and the usual supportive measures. All survived without complications. The eighth patient was treated by primary cardiorrhaphy and recovered. An additional patient, shot through the heart, failed to respond to aspiration of 7 cc. of the pericardial blood and was thought not to have a tamponade. Exploration revealed a through and through wound of the heart, penetrating the right pulmonary hilus as well. He died of ventricular fibrillation. A patient with a stab wound of the neck bleeding into the left thoracic cavity was subjected to immediate thoracotomy. A wound of the left auricle was successfully sutured. The cases are recorded in detail later in this article, and the available data in 4 instances are presented in graphic charts. No other patients with heart wounds were admitted to the hospital during this period. Cases of patients who died in the accident room, and therefore were not admitted to the hospital, are discussed separately.

We continue to be impressed by the simplicity and regularly successful outcome of pericardial aspiration. Aspiration is performed in the left fourth or fifth interspace parasternally with a 16 or 18 gage needle. The quantity of blood in the 7 cases reported varied widely, from 12 to 305 cc. (the latter amount being obtained in two aspirations, four minutes apart). In all these cases there was an unequivocal cardiac tamponade. In some of them, as in case 4 (J. R.), in which the venous pressure did not reach a normal level for several days (fig. 2), it can safely be assumed that aspiration removed only a portion of the blood from the pericardial cavity. A salient feature of the treatment is the striking rapidity of clinical improvement after removal of blood from the pericardium.

Despite the excitement attending the arrival of such patients, every effort is made to carry out repeated observations on the arterial and venous pressures. The latter, in particular, has a specific diagnostic and prognostic significance. In most cases we have not felt justified in withholding blood or plasma in order to observe the effect of aspiration alone. J. S. (case 9), however, entered the accident room cold, sweating, pulseless and wildly restless, with unobtainable blood pressure, inaudible heart sounds and no cardiac pulsation visible with the fluoroscope. Aspiration of 12 cc. of blood from his pericardial cavity trans-

7. Cooper, F. W., Jr.; Stead, E. A., Jr., and Warren, J. V.: The Beneficial Effect of Intravenous Infusions in Acute Pericardial Tamponade, ibid. **120**:822, 1944.

formed the clinical picture to one with blood pressure of 85 mm. systolic and 50 mm. diastolic (mercury), easily palpable pulse, audible heart sounds and cardiac pulsations plainly visible under the fluoroscope. Only then was the administration of blood plasma started. From a high point of 208 mm. of water prior to aspiration, the venous pressure fell gradually to 74 mm. of water in the next two and a half hours.

The usual clinical picture was that generally described. The standard signs of tamponade are low arterial pressure, elevated venous pressure, distant or absent heart sounds, decreased or absent cardiac pulsations as seen with the fluoroscope and the usual concomitant signs of shock. In addition, we find noteworthy the frequent occurrence of a relatively slow heart beat, when any is detectable, and the common occurrence of wild excitement in patients not completely prostrated. The electrocardiogram, even when taken in the first few minutes of treatment, has shown no specific change, except in case 11, in which the diagnosis of an injury of the coronary artery was made during life and demonstrated at autopsy. In several of the patients there subsequently developed some degree of hemothorax, which conceivably would indicate a leakage of pericardial fluid into the chest, preventing the delayed development of tamponade. It is particularly interesting that of the 8 cases, including case 11 (P. H.), in which tamponade was successfully treated by aspiration alone, 3 were of bullet wounds, 3 of knife wounds and only 2 were of ice pick wounds. The type of the weapon used to inflict the wound, therefore, need not affect the choice of treatment.

The possibility of cardiac tamponade is kept in mind in any case of a thoracic wound, particularly if it is precordial, and all such patients are closely watched. In case 1 signs of tamponade developed under observation during a period of almost two hours. Fluoroscopic examination and pericardial aspiration are the most helpful diagnostic measures. Arterial pressure and venous pressure should respond steadily after aspiration, and these, with the heart sounds and fluoroscopic findings, form the basis for evaluation of improvement. If improvement ceases or is not maintained at a satisfactory level, aspiration is repeated, as in case 8 (D. C.), in which 305 cc. of blood was aspirated in two attempts, 165 and 140 cc., respectively. If a second aspiration should fail to give relief, our plan is to take the patient to the operating room for cardiorrhaphy. The operating room is always alerted as soon as a patient with a wound of the heart is received in the accident department. It is our practice to take patients suspected of pericardial tamponade directly to the fluoroscopic room in the accident department and to perform all examination and treatment of the patient on the fluoroscopic table, with frequent observation of the heart. A needle is

at once placed in a peripheral vein, and administration of plasma is started prior to giving of whole blood. At times, dextrose injection in isotonic sodium chloride solution U. S. P. is injected preliminary to the administration of plasma. Administration of whole blood is continued until the patient's condition and blood pressure are stabilized at a satisfactory level. It would appear that in most cases of wounds of the heart which are not immediately fatal, bleeding is controlled and arrested by the tamponade and does not recur when the tamponade, which is now the life-threatening condition, is relieved by aspiration. Thus far we have seen no recurrence of symptoms of tamponade once the initial symptoms have been relieved.

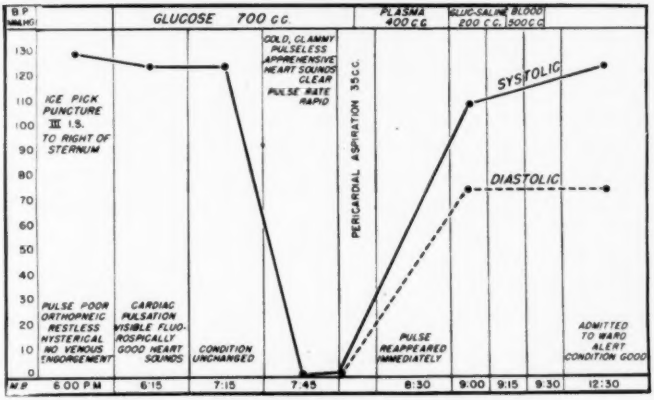

Fig. 1.—Graphic presentation of clinical data in case 1.

REPORT OF CASES

CASE 1.—M. B., a 17 year old Negro girl (fig. 1), was admitted to the accident room at 6 p. m. on July 16, 1942. She came to the hospital by car, upset and hysterical, stating that a short time before she had been stabbed in the chest with an ice pick. There was a small puncture wound in the third interspace on the right side beside the sternum. On admission, her blood pressure was 130 mm. systolic and 84 mm. diastolic. She had some discomfort when lying down, but no dyspnea. The heart sounds were of good quality, and cardiac pulsation was plainly visible fluoroscopically. There was no venous engorgement. Her condition remained unchanged for one hour and fifteen minutes of observation. Thirty minutes later she was heard moaning and was found to be cold, clammy and pulseless. Her heart rate was very rapid, but the sounds were of good quality. The arterial blood pressure could not be obtained. Fluids were given at once, and a pericardial aspiration, made in the fourth interspace on the left side, yielded 35 cc. of blood. A fairly good pulse returned at once, and the patient instantly felt greatly improved. She was given rapidly 500 cc. of plasma, 500 cc. of blood and

1,000 cc. of dextrose injection in isotonic sodium chloride solution U. S. P. Warmth quickly returned. Her blood pressure rose to 110 mm. of mercury systolic and 75 mm. diastolic and remained there. There was no evidence of either pneumothorax or hemothorax. An electrocardiogram, taken on July 17, was normal. For the duration of her one week stay in the hospital the patient was asymptomatic. She was last seen in December 1944, when she was thought to have schizophrenia, but otherwise appeared well.

Summary.—In a case of an ice pick wound of the heart, tamponade developed during observation and was relieved by aspiration of 35 cc. of blood from the pericardial cavity and the administration of 500 cc. of plasma, 500 cc. of blood and 1,000 cc. of dextrose injection in sodium chloride solution U. S. P.

CASE 2.—J. R., a Negro aged 20, was brought to the accident room at 7 a. m. on Feb. 14, 1943, fifteen minutes after having been shot in the chest. He was cold, pulseless and appeared to be moribund. There was a bullet wound in the third interspace to the left of the sternum. Aspiration of the pericardial contents yielded 7 cc. of blood, and faint heart sounds could be heard. The peripheral veins were not distended. The patient was thought to be in shock rather than to be suffering from tamponade, and unmatched group 0 blood was given at once. After the administration of 100 cc. of blood, his condition improved, and the blood transfusion was continued. The extremities became warm and the pulse palpable. The blood pressure rose to 100 mm. of mercury systolic and 70 mm. diastolic with continued transfusion, and he was sent to the ward. Coarse, bubbling rhonchi, heard all over the chest, suggested bleeding into the bronchial tree. The patient's condition remained static for about two hours, when his blood pressure again began to fall despite the administration of an additional 500 cc. of blood. With his blood pressure 80 mm. of mercury systolic and 20 mm. diastolic, he was taken to the operating room. He had thus far received 1,500 cc. of blood and 500 cc. of plasma. Although his general condition had improved greatly, he had never become conscious. The pericardium was exposed and had just been incised when the patient's pulse became imperceptible and the ventricles went into visible fibrillation. Neither administration of procaine hydrochloride, epinephrine and nikethamide (N,N-diethylpyridine-3-carboxamide) nor cardiac massage had any beneficial effect. A bullet wound of the right ventricle to the left of the pulmonary conus was sutured before the patient died, and there was observed another bullet wound, on the posterior surface of the right ventricle, which matched still a third hole in the pericardium on the right side, between the entrances of the venae cavae. It appeared that the latter wound probably relieved the tension of the potential cardiac tamponade and permitted the patient to survive as long as he did.

Summary.—In this case of perforating wound of the heart, the patient did not have tamponade. He died of ventricular fibrillation on the operating table.

CASE 3.—V. M., a Negro aged 21, was admitted to the accident room at 8:45 p. m. on March 24, 1944, about thirty minutes after he had been stabbed with a 3 inch (7.6 cm.) knife blade. He later told us that the blood had come out of the wound with great force and that he had walked a short distance and then collapsed. There was a stab wound in the fifth interspace just to the left of the sternum. He was pulseless; his blood pressure was unobtainable, and the heart

sounds were faint. On fluoroscopic examination, only faint cardiac pulsations were seen. The skin was cold and clammy, and the patient was perspiring profusely. He was irrational most of the time. Intravenous administration of fluids was started, but there was no improvement in his condition. Aspiration of the pericardial contents from below the xiphoid process yielded 15 cc. of blood. Fluoroscopic studies showed barely perceptible cardiac pulsations. Aspiration in the fifth interspace on the left side with a 16 gage needle yielded 100 cc. of dark blood, and the patient became quiet immediately. Pain in the calf of the left leg disappeared, and he fell asleep. The pulse became perceptible, and the blood pressure was 80 mm. of mercury systolic and 50 mm. diastolic at once. During the next hour, the blood pressure rose to 100 mm. of mercury systolic and 60 mm. diastolic, and a blood transfusion was given. Venous pressures taken in the accident room (time relation to therapy not stated) were 145, 165 and 175 mm. of water. The electrocardiogram on March 25 showed no abnormalities. Roentgenograms showed a possible small effusion in the left pleural cavity. The patient was discharged on April 3, to remain in bed for three weeks. He failed to keep his return appointment.

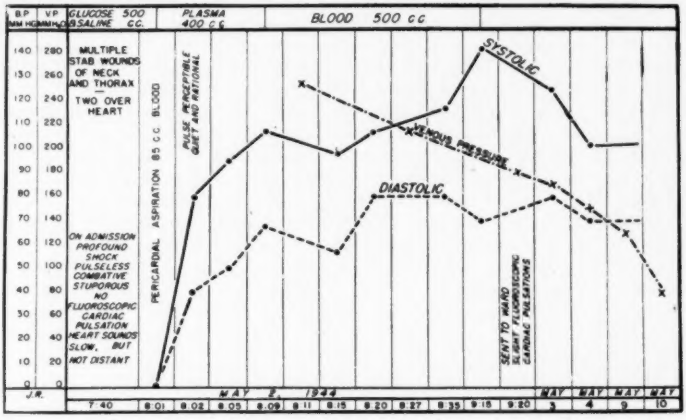

Fig. 2.—Graphic presentation of clinical data in case 4.

Summary.—This patient had tamponade from a stab wound. Aspiration of 115 cc. of blood produced dramatic relief, which was maintained by the administration of 500 cc. of blood.

CASE 4.—J. R., a Negro aged 22 (fig. 2), was admitted to the accident room in profound shock at 7:40 p. m. on May 2, 1944, twenty-five minutes after receiving multiple stab wounds over the thorax and neck. The wounds were not bleeding severely, and the clothes were not blood soaked. The veins in the neck were distended. Two of the stab wounds were over the precordium; one was over the sternum at the level of the fourth interspace and appeared to have passed to the right. The patient was pulseless, and his blood pressure was unobtainable. The heart sounds were slow, but not distant. An intravenous injection of dextrose was given instantly, followed almost at once by plasma. On fluoroscopic examina-

tion, no cardiac pulsations could be seen. The patient was still pulseless, semiconscious and combative. Aspiration yielded 85 cc. of blood from the pericardial cavity. His blood pressure immediately rose to 80 mm. of mercury systolic and 40 mm. diastolic, although it had been unobtainable immediately before aspiration. The patient became quiet and completely rational. The pulse was obtainable at the wrist. In the next hour and twenty minutes the blood pressure rose to 142 mm. of mercury systolic and 68 mm. diastolic and the pulse rate remained between 90 and 100. The first venous pressure reading, taken ten minutes after aspiration, was 253 mm. of water. Fifteen minutes later it was 213 mm. of water, and in another hour it had fallen to 185 mm. of water. By this time, an hour and twenty minutes after aspiration, fluoroscopic examination showed slight cardiac pulsations, and the patient was sent to the ward. Symptoms of cardiac tamponade had not recurred. The patient had received 400 cc. of plasma and 500 cc. of dextrose in saline solution by 8:05 p. m., at which time the administration of blood had been started. The pericardial aspiration was completed at 8:02 p. m., and the patient's blood pressure was then 80 mm. of mercury systolic and 40 mm. diastolic. By 8:45 p. m. 500 cc. of blood had been administered. His venous pressure was 170 mm. of water on May 3, 135 and 138 mm. of water on two observations on May 9 and 80 mm. of water on May 10. Roentgenograms of the chest showed no pleural fluid. Electrocardiograms, taken the following day, showed elevation of the S-T segments, which was construed as possibly representing pericarditis. These changes became less prominent in the electrocardiograms taken on May 5 and May 9. He was discharged from the hospital on May 12 with instructions to remain in bed for one week. He was last seen on May 19, apparently well.

Summary.—This patient had tamponade from a stab wound. Aspiration of 85 cc. of blood gave an immediate response. He received 500 cc. of blood, 400 cc. of blood plasma and 500 cc. of dextrose injection in isotonic sodium chloride solution U. S. P. His venous pressure continued to fall for several days, suggesting that some degree of tamponade had persisted.

CASE 5.—E. P., a 17 year old Negro youth, was brought to the accident room on Jan. 26, 1945, about ten minutes after he had been shot in the chest. At the time of admission he was pulseless, sweating profusely and agitated. The blood pressure was unobtainable. There was a bullet wound over the midsternum at the level of the second rib. Immediate fluoroscopic examination showed the bullet in the chest to the right of the midline about two interspaces below the point of entry. There were no visible cardiac pulsations. Slight venous distention was evident. Intravenous administration of dextrose injection in isotonic sodium chloride solution U. S. P. was started and immediately followed by injection of 250 cc. of plasma. At the end of twenty minutes the arterial blood pressure was obtained at 58 mm. of mercury systolic and 50 mm. diastolic, although the radial pulse was not palpable. Whole blood, 500 cc., was given at once. The venous pressure was then 195 mm. of water and rose in the next thirty minutes to 240 mm. of water. At this point pericardial aspiration was performed, and 160 cc. of blood was withdrawn. The radial pulse became obtainable at once, and cardiac pulsations became visible under the fluoroscope. The blood pressure was 72 mm. of mercury systolic and 54 mm. diastolic, and the pulse rate 125 when pericardial aspiration was performed and the blood transfusion started. Thirty minutes later the blood pressure was 114 mm. of mercury systolic and 76 mm. diastolic and the pulse rate 114. His condition being objectively and subjectively greatly improved, he

was transferred to the ward an hour and thirty minutes after he was first seen. The venous pressure was still 222 mm. of water. The next venous pressure, taken five hours later, was 145 mm. of water. His condition was stable thereafter. No electrocardiographic record was made at the time. Roentgenograms showed a moderate pleural effusion on the right. He was discharged fourteen days after admission. An electrocardiogram, taken on March 16, was recorded as normal. He was last seen in August and was still well.

Summary.—In a case of tamponade from a bullet wound, aspiration of 160 cc. of blood produced rapid improvement. The patient received 500 cc. of blood and 250 cc. of plasma. His venous pressure dropped slowly over a period of hours.

Case 6.—E. K., a Negro aged 42, was admitted to the accident room at 5:10 p. m. on July 5, 1945, fifteen minutes after having been stabbed with an ice pick. He was brought in by ambulance, prostrate, cold, sweaty and pale. There was a puncture wound in the fifth interspace, 1 cm. to the left of the midclavicular line. Fluids were administered intravenously and simultaneously 80 cc. of dark blood was withdrawn from the pericardium by aspiration. His blood pressure rose from 20 mm. of mercury systolic and 0 diastolic to 108 mm. of mercury systolic and 60 mm. diastolic and his venous pressure dropped from 250 to 140 mm. of water. His general condition improved remarkably, and he became responsive and cooperative. The response, as shown by the change in venous pressure and in his general condition, was almost immediate. At this point a transfusion of whole blood was started, and he was sent to the ward. No notation was made of cardiac pulsation. The electrocardiogram on July 14 was normal. On July 18 the roentgenogram for the first time showed fluid in the left side of the chest, and in the next two days a total of 575 cc. of pink fluid was removed. He was discharged on July 20, having been allowed out of bed for the first time on July 9. He was not seen after discharge.

Summary.—In a case of tamponade from an ice pick wound, aspiration of 80 cc. of blood produced almost instantaneous return to normal levels of all indexes to his condition, after which 500 cc. of blood was given.

Case 7.—W. A. P., a Negro aged 44, entered the accident room at 6 p. m. on Feb. 24, 1946, fifteen minutes after he had received multiple stab wounds of the chest and abdomen. He was comatose, without discernible pulse or blood pressure. There were two stab wounds in the fourth or fifth interspace just to the left of the sternum, several stab wounds elsewhere and a deep stab wound in the left flank. Heart sounds were inaudible. Administration of blood was started in the accident room, and the patient was taken at once to the operating room. Incision was made at 7 p. m., the heart being exposed transpleurally. A stab wound at the base of the right ventricle was bleeding briskly, and the pericardium was filled with clotted blood. With this blood evacuated and with the finger held in the heart wound, the pulse became strong and the blood pressure rose to 90 mm. of mercury systolic. The heart wound was sutured, and the thoracic wall closed. The abdomen was explored, and no visceral injury was found. The patient convalesced satisfactorily. Electrocardiograms on February 25 and March 14 were read as normal. The patient was discharged by ambulance on March 15, to remain in bed at home. He was last seen on April 22, apparently well.

Summary.—In a case of tamponade from a stab wound, primary cardiorrhaphy was followed by recovery.

CASE 8.—D. C., a Negro, aged 39 (fig. 3), entered the accident room at 10:55 p. m. on Jan. 14, 1947. He was pulseless, cold, sweating, combative and stuporous, fifteen to twenty minutes after he had been stabbed with a knife. There was a stab wound in the second interspace 2 cm. to the left of the sternum. The heart sounds were inaudible; respirations were rapid. The peripheral veins were moderately distended. Immediate fluoroscopic examination showed no visible cardiac pulsations. Pericardial aspiration through the fourth interspace on the left yielded 165 cc. of blood. The pulse appeared at the wrist for three or four minutes, and the heart sounds become barely audible. The venous pressure at 11:11 p. m. was 280 mm. of water. Plasma, 500 cc., was given intravenously. Another pericardial aspiration yielded 140 cc. of blood, a total of 305 cc. within sixteen minutes. Almost instantly the radial pulse was perceptible and strong, and cardiac pulsations

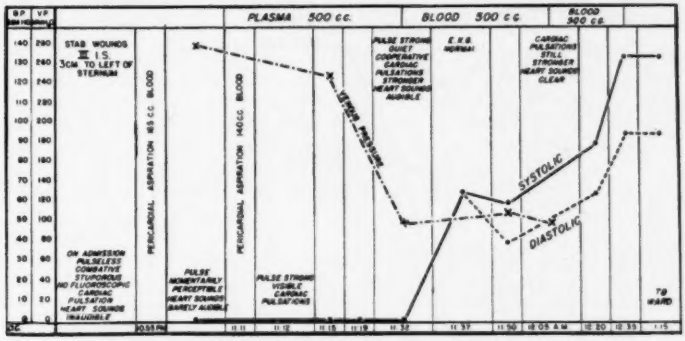
PLASMA 500 c.c.
BLOOD 500 c.c.
BLOOD 500 c.c.
STAB WOUNDS
II I.S.
3CM. TO LEFT OF STERNUM
PERICARDIAL ASPIRATION 165 C.C. BLOOD
PERICARDIAL ASPIRATION 140 C.C. BLOOD
PULSE STRONG QUIET COMPARATIVE CARDIAC PULSATIONS STRONGER HEART SOUNDS AUDIBLE
E.K.G. NORMAL
CARDIAC PULSATIONS STILL STRONGER HEART SOUNDS CLEAR
STRONG PULSE
SYSTOLIC
DIASTOLIC
ON ADMISSION PULSELESS COMBATIVE STUPOROUS NO FLUOROSCOPIC CARDIAC PULSATION HEART SOUNDS INAUDIBLE
PULSE MOMENTARILY PERCEPTIBLE HEART SOUNDS BARELY AUDIBLE
PULSE STRONG VISIBLE CARDIAC PULSATIONS
TO WARD

Fig. 3.—Graphic presentation of clinical data in case 8.

became visible on fluoroscopic examination. At 11:15 p. m. the venous pressure was 250 mm. of water. At 11:19 p. m. the pulse was 80 per minute; the blood pressure was still unobtainable. At 11:32 p. m., the extremities were dry, the pulse was strong and full and the patient was quiet. Fluoroscopic examination showed stronger cardiac pulsations, and the heart sounds were audible. The venous pressure was now 100 mm. of water, and the pulse rate, 90. The patient had received 500 cc. of plasma, which was now followed with 500 cc. of blood. At 11:37 p. m. his blood pressure was obtainable at 65 mm. of mercury systolic, and the respiratory rate was 26. The patient was quiet, rational and cooperative. It was remarked that from the beginning the heart had always appeared large under the fluoroscope, both to the right and to the left. An electrocardiogram was taken at 11:40 p. m. At 11:50 p. m. the pulse was 72, the venous pressure 110 mm. of water and the blood pressure 60 mm. of mercury systolic and 40 mm. diastolic. At 12:05 a. m., the patient was warm and dry, the cardiac pulsations were continuing to improve, the pulse was 62, the sounds at the apex were clear and the venous pressure was 100 mm. of water. The patient had received 500 cc. of blood, and administration of another 500 cc. was started. The systolic blood pressure was then 80 mm. of mercury. It

rose steadily, and at 12:35 the blood pressure was 135 mm. of mercury systolic and 95 mm. diastolic, the second 500 cc. of blood having been administered. The patient was admitted to the ward at 1:15 a. m. with a pulse rate of 80, a respiratory rate of 24 and a blood pressure of 135 mm. of mercury systolic and 90 mm. diastolic. During the next twenty-four hours his blood pressure steadily rose to a frankly hypertensive level, with the systolic pressure ranging from 150 to 170 mm. and the diastolic from 110 to 130 mm. From the time of his admission to the ward he improved steadily, with no return of the symptoms of tamponade. Roentgenograms of the chest failed to confirm the fluoroscopic impression of cardiac enlargement, but in the ward he showed consistent hypertension. The original electrocardiogram and two subsequent ones on January 17 and 27 were read as normal. The patient was up in a wheel chair on January 28 and was discharged on January 30. He was last seen in the outpatient department on February 3, when he stated that he had decided not to remain at rest, as instructed, and had resumed full activity. He was asymptomatic.

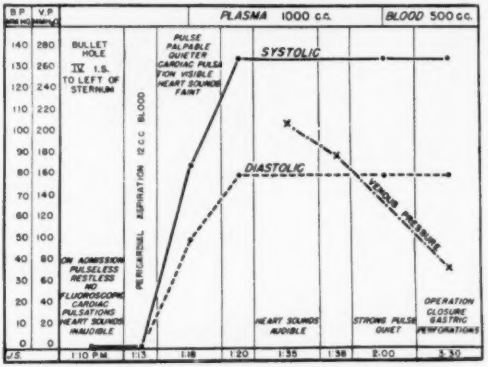

Fig. 4.—Graphic presentation of clinical data in case 9.

Summary.—In a case of tamponade from a stab wound, a total of 305 cc. of blood was aspirated from the pericardium over a period of sixteen minutes, at the end of which time improvement was rapid. The patient received 500 cc. of plasma and 1,000 cc. of blood.

CASE 9.—J. S., a Negro aged 29, with a long history of previous stab and bullet wounds was admitted to the accident room (fig. 4) at 1 p. m. on Feb. 12, 1947, fifteen minutes after having been shot in the chest. There was a small bullet hole in the fourth interspace on the left. He was cold, sweating, pulseless and wildly restless, but conscious and fairly cooperative. The blood pressure was not obtainable; under the fluoroscope, the heart was not seen to pulsate. Heart sounds were inaudible. Aspiration of 12 cc. of blood with a no. 18 needle through the fourth interspace to the left of the sternum produced dramatic relief. The pulse returned immediately, and cardiac pulsations became plainly visible under the fluoroscope, although the heart sounds still were not clear. The blood pressure rose at once to 85 mm. of mercury systolic and 50 mm. diastolic, and intravenous administration of plasma was begun. A .22 caliber bullet could be seen below

the left side of the diaphragm. By 1:20 p. m., his blood pressure was 135 mm. of mercury systolic and 80 mm. diastolic. At 1:38 the venous pressure reading was 180 mm. of water. By 2:00 p. m., he had received 1,000 cc. of plasma; his pulse was strong, and he was warm, dry and quiet. An electrocardiogram, taken at 2:15 p. m., showed changes in the T waves in precordial leads, interpreted as suggestive of myocardial damage in the region of the anterior part of the septum (inversion in CF_1 and CF_2 leads and diphasic in the CF_3 lead). His general condition remained unchanged, the arterial blood pressure holding at 135 mm. of mercury systolic and 80 diastolic and the venous pressure dropping to 74 mm. of water by 3:30 p. m. He was then taken to the operating room, where a through and through wound of the stomach was repaired. Little blood or soiling was found in the peritoneal cavity. After operation a moderate amount of fluid collected in the left pleural cavity. Convalescence was smooth, and he was discharged on February 23.

Summary.—In a case of cardiac tamponade from a bullet wound, aspiration of 12 cc. of blood produced immediate and dramatic response, after which 1,000 cc. of plasma was given.

CASE 10.—T. C., a Negro aged 37, was brought to the accident room on June 29, 1947, about thirty-five minutes after being stabbed in the neck, back and arms. He was conscious but in shock, with a blood pressure of 60 mm. of mercury systolic and 40 mm. diastolic. There were two stab wounds at the base of the neck on the left. There was no external evidence of bleeding, but the left side of the chest was dull to percussion. It was thought that a wound of the great vessels was bleeding into the left side of the chest. The patient was given 1,000 cc. of plasma and 500 cc. of blood as preparations were made for the operation.

The chest was opened in the second interspace on the left side. A laceration 2 cm. in length in the left auricle was bleeding freely into the left side of the chest. The laceration was sutured while 3,500 cc. of blood was administered. The patient's blood pressure was unobtainable at several points in the procedure, but he was returned to the ward with a blood pressure of 138 mm. of mercury systolic and 80 mm. diastolic. He made an uneventful recovery. Electrocardiograms on June 30 and July 7, 9 and 11 showed low T waves and elevation of the S-T segments, changes which were interpreted as indicative of pericarditis. By July 15, prominent Q waves in lead II and large Q waves in lead III had developed. The patient was discharged on July 17, after having been up for four days.

Summary.—The patient had hemothorax from a stab wound of the left auricle, with the wound of entry in the neck. Cardiorrhaphy was followed by recovery. Immediate operation is the treatment of choice in such a case.

The 10 patients whose records have been presented were the only ones with heart wounds who were admitted to the hospital in the period under study. We have investigated the records of all patients pronounced dead in the accident room during the period in which the 10 patients whose records have just been described were seen. Among these were 31 patients with penetrating thoracic wounds, 20 of whom were dead on arrival at the hosiptal. Of the remaining 11 patients,

6 were found not to have wounds of the heart. This leaves 5 patients with wounds of the heart who died in the accident room and who are of interest in connection with the present report. The pertinent data in these 5 cases are presented in the table. It may be observed that

Clinical Data on Five Patients Who Died of Wounds of the Heart.

Accident Room Record

Patient	Interval from Injury to Admission, Min.	Interval from Admission to Death, Min.	Wounds	Clinical Impression	Treatment
J. C.....	40	5	Stab, wound, right side of chest	Hemothorax, right; cardiac tamponade?	Intravenous administration of fluids; intracardiac injection of nikethamide; aspiration of 60 cc. blood from pericardium
J. H.....	15	5	Gunshot wound left side of chest posteriorly	Gunshot wound left side of chest	Died as intravenous injections of fluids and nikethamide were started
W. D.....	5	5	Stab wound left left side of chest	Cardiac tamponade; massive hemorrhage	Intravenous injection of fluids and plasma; nikethamide intracardially
E. S.....	Not known	55	Multiple stab wounds, chest	No penetrating wound of thorax	Plasma 1,300 cc. in 40 min.; pericardial aspiration of 8 cc. of blood; blood pressure 70/0 mm. Hg after 50 min.; venous pressure 270 mm. H_2O
P. H.....	Not known	120	Bullet wound left side of chest	Hemopericardium; injury to coronary artery (EKG)	Aspiration of 175 cc. of blood; 1,000 cc. of plasma and 300 cc. of blood administered intravenously; venous pressure then began to rise and patient died of cardiac failure

Medical Examiner's Report

Patient	Viscera Injured	Internal Loss of Blood	Heart Wound	Hemopericardium	Evaluation
J. C.....	Right lung, heart	2,500 cc. right pleural cavity	Right auricle	Not mentioned	Died of hemorrhage
J. H.....	Left lung, heart	750 cc. left pleural cavity; 375 cc. right pleural cavity	Left ventricle and interventricular septum	120 cc.	Died almost at once, probably of tamponade
W. D.....	Heart	2,500 cc. left pleural cavity	Apex of left ventricle	Not mentioned	Died of massive hemorrhage
E. S.....	Heart	1,250 cc. left pleural cavity	Pulmonary conus	90 cc. removed	Died of hemorrhage and tamponade; might have been saved had diagnosis been made on admission
P. H.....	Left lung, heart	800 cc. left pleural cavity	Myocardium of base of left ventricle; left anterior descending coronary artery	None removed	Died of heart failure after recovery from tamponade; missile injured coronary artery and caused infarct

J. C. had a stab wound in the right auricle and that there was bleeding into the right side of the pleural cavity. He was brought to the accident room forty minutes after injury and died almost at once. J. H. had a gunshot wound in the left ventricle and in the interventricular septum, with accompanying hemopericardium, and died almost as soon as he was

seen. W. D. died, almost at once of massive hemorrhage into the left side of the chest from a wound in the left ventricle. E. S. might have been saved by appropriate therapy. Autopsy revealed a wound of the pulmonary conus, 90 cc. of blood in the pericardium and 1,250 cc. in the left pleural cavity. His survival period in the accident room was fifty-five minutes, long enough to have permitted adequate aspiration therapy or operation. However, he had multiple stab wounds, none of which were thought to be penetrating. P. H. is of special interest; therefore his report follows in detail.

Case 11.—P. H., a Negro aged 19, was brought into the accident room at 11:40 p. m. on May 24, 1947, shortly after having been shot in the left side of the chest. He was unconscious, cold and sweating. No pulse could be detected at the wrist. The heart sounds were inaudible, and cardiac pulsations were not visible under the fluoroscope. The blood pressure could not be obtained, but a feeble femoral pulse with a rate of 88 was detected on palpation. There was moderate venous distention. Administration of plasma was begun at once, and aspiration of the pericardial contents was made through the fifth interspace on the left side. After 80 cc. of blood had been removed, it was possible to hear the heart sounds, and his systolic blood pressure was 40 mm. of mercury ten minutes after admission. At 11:55, his venous pressure was 170 mm. of water; 500 cc. of plasma had been given, and a second injection of 500 cc. was begun. Fluoroscopic examination showed evidence of a small hemothorax on the left and a high, fixed diaphragm on the same side suggested injury to the phrenic nerve. At midnight, the cardioscope showed characteristic changes of a left anterior infarction. At 12:05 a. m, another aspiration was begun, and in three minutes an additional 95 cc. of blood was removed from the pericardium. At the beginning of this aspiration the venous pressure was 145 mm. of water, and at the end it was 128 mm. The patient became conscious, rational and interested in his condition. His previous history disclosed that in 1944, at the age of 15, he had been a patient in the Johns Hopkins hospital, where the diagnosis was combined mitral and aortic valvular disease. He stated that he had not recently been dyspneic. At 12:13 a. m. the venous pressure was 140 mm. of water and the blood pressure 70 mm. of mercury systolic and 44 mm. diastolic, and at 12:22 a. m., the venous pressure was 135 mm. of water and the arterial blood pressure 72 mm. of mercury systolic and 46 mm. diastolic. His pulse was strong with a rate of 96; the skin was warm and dry. He had had the second 500 cc. of plasma, and a transfusion of whole blood was begun. At 12:31, he had had about 200 cc. of blood; his blood pressure was 75 mm. of mercury systolic and 40 mm. diastolic, but the venous pressure had risen to 165 mm. of water. It occurred to us that his large rheumatic heart, further handicapped by the coronary infarction which the medical consultant (Dr. Witten) diagnosed, might have been overtaxed and failure of the right side of the heart was beginning. Accordingly, the rate of administration of the blood was greatly decreased, and the venous pressure then dropped for a time, being 158 mm. of water at 12:38 p. m. and 150 mm. of water at 12:39 p. m. At the same time, his blood pressure rose to 82 mm. of mercury systolic and 54 diastolic, and his face became warm and dry. In the next twenty minutes his venous pressure rose to 170 to 180 mm. of water, although his arterial blood pressure remained up. At 1:00 a. m. the arterial pressure was 84 mm. of mercury systolic and 54 mm. diastolic and his venous pressure 165 mm. of water. He had had 300 cc. of blood, when the transfusion was stopped. We thought that his recovery was assured and that he had some

blood in the pericardium which would be absorbed or would leak into the left side of the thorax. We felt that there might still be a moderate degree of tamponade and that there was probably injury of the left coronary artery, and perhaps of the phrenic nerve.

At 1: 10 a. m., he was rational; his skin was warm and dry; his blood pressure was 100 mm. of mercury systolic and 60 mm. diastolic, his pulse was strong, and his heart sounds were clear. There was great difficulty in getting the venous pressure, which appeared to be incredibly elevated on each determination. At 1: 21 a. m., the dehydrocholic acid ("decholin") circulation time was 8 seconds. At 1: 30 a. m. the venous pressure was 240 mm. of water. This seemed so high that the test was repeated, but each successive determination gave a higher reading. In view of his good general condition, this was erroneously attributed to a technical error. Suddenly the patient gasped, rolled his eyes up, became pulseless, and, after a few gasps, when blood foamed at the nose and mouth, he died. Postmortem taps of both pleural cavities and of the pericardium yielded no blood.

We thought that the sudden death was due to cardiac failure from the coronary infarction.

Medical Examiner's Report.—Autopsy revealed 800 cc. of blood in the left pleural cavity, 200 cc. of clear transudate in the right pleural cavity and about 5 cc. of clotted blood in the pericardium. The right lung was edematous and heavy, weighing 700 Gm. and being filled with fluid, which bubbled out the bronchi. The left lung was atelectatic, and a bullet hole passed through the lower portion of the upper lobe. The heart seemed enlarged. There was a through and through wound of the myocardium of the left ventricle at the base, close to the coronary artery and not entering the chamber of the heart. The bullet was lodged in the sternum. The missile had transected the first branch of the anterior descending branch of the left coronary artery and produced a large hematoma in the myocardium and epicardium which appeared to have compressed the anterior descending branch of the left coronary artery. The myocardium in this area was whitish and dull, and its appearance was compatible with that of a fresh infarction. There was little evidence of rheumatic heart disease despite the previous history, but there was a fresh, fibrinous vegetation, 8 mm. in size, on the mitral valve. This appeared to be an active vegetation and was a purely coincidental finding. There was no right ventricular hypertrophy.

Comment.—This man recovered from his tamponade as a result of aspiration of 175 cc. of blood from the pericardium and died of cardiac failure due to injury to the coronary artery and traumatic infarction, probably hastened by intravenous therapy (1,000 cc. of plasma and 300 cc. of blood being given in seventy-five minutes). He did not require operative treatment, since the missile had not entered the heart chamber, and it may be said that his chance of recovery would not have been enhanced by operation. The diagnosis of infarction of the left anterior wall of the heart was made from the cardioscopic examination twenty minutes after the patient was first seen and should perhaps have been a warning against vigorous intravenous therapy.

Summary.—In this case, of pericardial tamponade due to a bullet wound, there was rapid response to aspiration of 175 cc. of blood. Death was due to pulmonary edema after 1,000 cc. of plasma and

300 cc. of blood had been administered. Autopsy showed no residual hemopericardium but revealed injury by the bullet to the anterior descending branch of the left coronary artery.

SUMMARY

Seven consecutive patients with pericardial tamponade due to hemopericardium were successfully treated by aspiration alone; 1 patient was successfully treated by immediate cardiorrhaphy, and 1 patient without tamponade died as a result of a perforating bullet wound of the heart and pulmonary hilus. One patient bleeding into the left side of the chest from a laceration of the left auricle was successfully treated by suture of the auricle. One patient with cardiac tamponade due to a bullet wound responded well to aspiration, only to die abruptly two hours after he was first seen. Autopsy showed division of the anterior descending branch of the left coronary artery, fresh infarction of the left ventricle and evidence of cardiac failure with pulmonary edema. Three additional patients died of heart wounds within five minutes of being admitted to the accident room. A fourth lived fifty-five minutes and might have been saved, but was thought not to have a penetrating wound.

Pericardial aspiration is the most important initial treatment for cardiac tamponade due to wounds of the heart. Facilities for open operation should be held in readiness, but it appears that with most wounds which cause cardiac tamponade, and are not immediately fatal, aspiration alone will suffice. Administration of blood and plasma is an important adjuvant.

NOTE—Since this report was submitted, 2 more patients have been treated for wounds of the heart. Both had tamponade, and both recovered.

The first patient, A. S., had a stab wound of the heart. Four pericardial aspirations over a space of fifty-five minutes yielded 773 cc. of blood, with dramatic recovery from a moribund state.

The second patient, C. C., had tamponade from a bullet wound. After 653 cc. of blood had been removed from the pericardium, in four aspirations, the patient was sent to the operating room for exploration of an abdominal bullet wound. His blood pressure was then 155 mm. of mercury systolic and 95 mm. diastolic, while the venous pressure was 230 mm. of water. With induction of anesthesia, arterial pressure fell sharply and venous pressure rose. Therefore, the pericardium, was aspirated of another 120 cc. of blood and a thoracotomy performed. There was a myocardial "increase," with one spurting vessel easily controlled by sutures. The abdomen was then explored, and perforations of the stomach were closed. He made a good recovery.

Johns Hopkins Hospital.

TETANUS TREATED AS A RESPIRATORY PROBLEM

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AND
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THE RECOGNITION of the importance of respiratory obstruction due to tracheal and pharyngeal secretion in certain conditions and the ability to control it have led to a completely changed prognosis in many diseases. This has been true especially in bulbar poliomyelitis.[1] During the year 1947 no patient with anterior poliomyelitis died at the Evanston Hospital although there were 60 cases, 7 of which were of the bulbar type. Five of these required tracheotomy. One patient with myasthenia gravis, accumulation of throat secretions and impending asphyxia was treated on the same basis, with rapid relief.

Certain other states would seem to have the same common denominator of respiratory obstruction, anoxia, respiratory weakness and danger of drowning from aspirated secretion. We thought that tetanus, with convulsions under control by curare, would present much the same picture and respond to the same measures. When a patient with severe tetanus came under our care we found in fact that she did respond to the treatment on this basis so remarkably that we believe it could be applied to most cases of severe tetanus with satisfactory results. The treatment was found to be relatively simple and seemed in retrospect to be so logical and dependable that it should be adaptable to most serious infections with tetanus.

Curare has been used in the treatment of tetanus since 1894, as reported by Cullen.[2] Tracheotomy was advised for grave emergencies by Spaeth.[3] Isacson and Swenson[4] used a respirator with apparent benefit but without recovery in 1 case of tetanus. Tracheotomy was not done. So far we have not found in the literature that the three measures have been combined in any case. This, we believe, may be necessary for the reasons to be discussed.

From Northwestern University School of Medicine and Evanston Hospital.

1. Galloway, T. C.: Management of Respiratory Complications of Poliomyelitis, Arch. Otolaryng. **46**:125-136 (Aug.) 1947.

2. Cullen, S. C., and Quinn, S. C.: Use of Curare in the Treatment of Tetanus, Surgery **14**:256, 1943.

3. Spaeth, R.: Tetanus, in Tice, F.: Practice of Medicine, Hagerstown, Md., W. F. Prior Company, Inc., 1924, vol. 5.

4. Isacson, S. E., and Swenson, S. A.: Curare in the Treatment of Tetanus, Nebraska M. J. **26**:136, 1941.

In bulbar poliomyelitis it is found that if the airway is cleared and kept clear by aspiration, postural drainage and, when necessary, tracheotomy or the use of the respirator or both most patients will recover. The improvement may be so rapid—a matter of minutes at times—that it indicates that the associated depression is much more likely to have been secondary to anoxia (or carbon dioxide accumulation) of peripheral respiratory origin than to infection. It seems also definitely to be indicated that in a patient in whom respiratory weakness, paralysis or drug action makes it necessary to use a respirator, if the secretion cannot be positively and constantly eliminated, tracheotomy must first be done. Why this is so was shown previously in a discussion of poliomyelitis.[1]

REPORT OF A CASE

On July 2, 1946, operations were performed on both knees of a 17 year old girl for recurrent congenital dislocation of the patellae. The operative wounds healed without difficulty, and the patient returned to full activity. Late in February 1947 a small ulcer developed in the proximal end of the operative scar on the left side. This developed into cellulitis of the thigh. The patient was admitted to the hospital at that time, and the inflammation subsided under complete bed rest, elevation of the extremity, hot compresses and administration of penicillin. Culture of material from the wound showed hemolytic staphylococci and hemolytic streptococci. She was discharged from the hospital wearing a small dressing over the nearly healed ulcer.

She was seen again on March 25, 1947, with an acute flare-up of the inflammation about the proximal end of the incision and with a small ulcer in the scar. She was again hospitalized and treated as at the time of the previous admission. Material from the wound was cultured, and it was inspected with a gloved finger. It was about 2.5 cm. in diameter and 2 cm. deep. The walls were smooth, and there were no pockets. Three days after admission symptoms of tetanus developed. After recovery, when she could be questioned, the patient stated that she had gone horseback riding ten days prior to the last admission to the hospital, and this may represent the time of introduction of the organisms into the wound.

Material from the wound was cultured on two occasions, and the tetanus bacilli were not found. However, the organisms are difficult to culture from such a wound, and there can be little doubt that the diagnosis of tetanus was correct, because the clinical manifestations were so marked and typical. The symptoms included an elevation of temperature, rigidity of the back and neck with opisthotonos, trismus, frothing at the mouth, difficulty in breathing, difficulty in swallowing, inability to open the mouth, production of stringy, frothy viscous secretions and acute, generalized tonic spasms or convulsions.

As soon as the diagnosis of tetanus was made, the patient was given 80,000 units of tetanus antitoxin intravenously and 20,000 units locally about the ulcer. Luminal sodium® (phenobarbital) was used for sedation, but because the spasms became increasingly severe she was given 60 units of curare, in the form of intocostrin,® intravenously. Administration of oxygen, 6 liters per minute by nasal catheter, was started. At the time the curare was given the stimulus of inserting a needle set off a tonic convulsion, during which the opisthotonos became extreme and respirations ceased for a half-minute at a time because of the spasm of the abdomen and chest. The effect of the intravenous administration of curare was dramatic: the opisthotonos decreased until it was possible for the patient to lie flat on the table; the jaw became relaxed, as did the skeletal muscles, and there was a release of the spasm of the respiratory muscles, so that more normal breathing was possible. However, when the effect of the curare was at its height, flaccid respiratory paralysis was noted, and it was necessary to insert an intratracheal tube and to give artificial respiration by bag for a period of several minutes. This flaccid paralysis was believed to be due to the use of curare.

When we found that the convulsive seizures could be controlled so readily by the use of curare, an attempt was made to set up a balanced system whereby the patient received a constant dosage sufficient to permit relaxation of muscles and aeration but not enough to cause respiratory paralysis. A continuous intravenous drip was started to provide adequate fluid intake. To each liter of fluid was added 10,000 units of tetanus antitoxin and up to 300 units of curare in the form of either *d*-tubocurarine chloride solution or intocostrin® (E. R. Squibb & Sons). An insufficient quantity of *d*-tubocurarine was available for continuous use, and intocostrin® was used interchangeably with it. It was necessary to give an occasional booster dose of curare intravenously or intramuscularly. We attempted to give slightly less than the amount required to keep the patient below the threshold of convulsions by the intravenous drip because of the danger of overdosage of curare and depended on close observation of the patient for the amount and timing of booster doses.[5]

After thirty-six hours of this management it was realized that a narrow zone existed between the flaccid respiratory paralysis caused by curare and the considerable respiratory embarrassment caused by the recurrent, extreme, tonic muscle spasm of the trunk characteristic of

5. The administration of the curare and the aspiration of secretions from the trachea and pharynx and artificial respiration were done by members of the department of anesthesiology when necessary, and especial credit for this patient's recovery must be given to Dr. Carl Johnson and Dr. Dorothy Darling, who alternated in constant attendance for the first seventy-two hours.

tetanus. In addition, the collection of viscid secretions in the respiratory passages added a mechanical obstacle to the free exchange of gases. Postural drainage of at least 25 degrees was difficult to maintain and relatively inefficient; aspiration with a catheter and a motor-driven suction apparatus frequently caused tonic convulsions which stopped respiration unless the patient was completely curarized. The use of atropine was tried, but if it had any effect on cutting down the quantity of secretions, it made them more stringy and sticky, so that the total effect was to make breathing more difficult. Moreover, the pulse became somewhat more rapid at times, and it was apparent that the patient was tiring and was obtaining insufficient gaseous exchange.

It was then reasoned, with subsequent justification in practice, that a tracheotomy and the use of a respirator would make possible the maintenance of a clear air passage and positive control of air exchange. With the tracheotomy, the thick secretion in the pharynx and larynx would be by-passed and the recurring laryngeal spasms would be of no consequence. It would also permit direct removal of the secretions in the trachea. The use of a respirator would make possible more latitude in the dose of curare. It was recognized that if a respirator were used without tracheotomy, secretion would be drawn into the lower airway and the pulmonary field. This could result in drowning the patient in her own secretions and produce atelectasis with secondary pneumonitis and sequelae, which have been so commmonly found in patients dying of tetanus. It seemed, moreover, that when the patient was fully curarized the clinical picture was similar to that seen in cases of bulbospinal paralysis, in which the use of the respirator proved effective after tracheotomy.

Once decided on, the procedure did not prove difficult. The patient was placed on the bed of the opened respirator in order to provide a quick change over after tracheotomy. The anesthetic tracheal rubber tube was in place, connected to the oxygen bag with which intermittent artificial respiration was being done by the anesthetist. The tube aided in fixing and raising the trachea. It was cut down on, an oval section of cartilage being removed from the third tracheal ring rather than at the usual lower level, so that it would lie above the respirator collar. A no. 4 tracheotomy tube was inserted, the wound packed and the oxygen tube quickly attached by a close-fitting rubber tube to the cannula.

When the patient's head was slid through the neck opening of the respirator collar, the respirator quickly closed, and the patient was immediately in good coordination with the machine. Her color improved, and the pulse slowed and became of good quality. It seemed much easier for her to rest and to sleep.

The rubber collar was satisfactorily held away from the tracheotomy tube by a soft "gray iron" bar ⅜ (0.96 cm.) by ¾ inch (1.91 cm.) wide and 10 inches (25.4 cm.) long, bent at a suitable angle by a wrench and fixed under the steel flange around the collar.

The respirator was maintained in postural drainage of about 25 degrees and secretion aspirated through the tube when there was any sign of obstruction. Because a good mechanical humidifier was not available, there was some drying of secretion. Crusts and plugs were easily removed by aspiration after irrigation with several droppers of warmed 3 per cent solution of sodium bicarbonate followed by isotonic sodium chloride solution. At times the tube was removed and deeper aspiration made directly through the wound. The patient was well synchronized with the respirator when fully curarized. At levels at which some spasm or twitching was provoked by manipulation, the respirator was effective, although there then appeared to be ectopic respiratory efforts outside the regular cycle.

It is stated that prolonged curarization may produce a state similar to surgical shock and that the use of the drug must therefore be intermittent.[6] The patient did at times have a rapid and irregular pulse, but there was no circulatory collapse. It was not easy to take her blood pressure when she was in the respirator, and this was not done.

It may be that the reason she did not show the picture of shock is that the amount of curare given via the continuous intravenous drip was not sufficient to produce complete flaccid paralysis. In fact, we attempted to err on the side of giving too little curare by this route, so that occasional booster doses were necessary. There was always greater than normal muscular tone; twitching and jumping of muscles could be observed at almost any time.

After ten days the dosage of curare was gradually diminished, and after twelve days it was discontinued. The use of the respirator was stopped for increasingly longer intervals. Even after the patient could breathe well voluntarily she was much more comfortable in the respirator for several days, or until she had regained her strength and had got back her normal respiratory habit.

Fluid and electrolyte balance, protein and vitamin needs were easily maintained by the intravenous route for the period of curarization. After the fifth day feeding by gavage was satisfactory through a no. 16 nasal catheter, which was kept closed when food was not being administered, in order to prevent indrawing of air. The respirator was left flat for one half hour after each feeding.

6. Cullen, S. C.: Curare, Its Past and Present, Anesthesiology **8**:478-488 (Sept.) 1947. Adrian, J., and Ochsner, A.: Some Observations on the Use of Curare in the Treatment of Tetanus, Surgery **22**:509-515 (Sept.) 1947. Curare in Clinical Medicine, E. R. Squibb and Sons, November 1948.

The dosage of curare was determined on the basis of the patient's reaction only. The largest amount given in any one twenty-four hour period was 1,500 units, and the total amount over a twelve day period was 11,750 units. The total number of units of tetanus antitoxin was 440,000. Administration of penicillin was continued throughout the period of treatment.

On the nineteenth day after the onset of symptoms the patient was up in a wheelchair, and on the twenty-third day all medication was discontinued.

Neurologic examination prior to the patient's discharge from the hospital showed an entirely normal condition, and there appeared to be no personality or intellectual change. An electrocardiogram was normal. The wound was nearly healed. Physical examination revealed no abnormalities.

SUMMARY

The combination of three measures—curare by continuous intravenous drip, tracheotomy and respirator—has been described in a severe case of tetanus and seemed to have these definite advantages:

1. It changed a hazardous treatment requiring unremitting care to a relatively simple, manageable, easily standardized procedure.

2. It allowed the use of curare in a latitude of dosage that is comparatively safe.

3. It guarded against dangerous periods of anoxia.

4. It by-passed secretions in the upper respiratory passage and permited aspiration of secretions in the lower airway; it avoided atelectasis and secondary pulmonary infections due to obstruction.

ACUTE PANCREATITIS AND ITS SEQUELAE

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THE TERM acute pancreatitis calls to the mind of most surgeons the relatively uncommon highly fatal acute hemorrhagic form of the disease. To Elman [1] must go much of the credit for pointing out the frequency with which the disease occurs in milder form and for emphasizing the necessity for routine determinations of serum amylase in all cases of pain in the upper abdominal area.

The clinical manifestations of the disease vary greatly. In acute hemorrhagic necrosis the attack is ushered in by the sudden onset of agonizing pain in the upper abdominal area, frequently radiating through to the back, accompanied with protracted nausea and vomiting. The patient is prostrated, the pulse weak and thready and examination reveals tenderness and spasm across the upper part of the abdomen. A sense of fulness in the epigastrium is frequently noted, or a mass consisting of the greatly swollen pancreas may be felt. A peculiar grayish cyanosis is sometimes observed.

From this extreme, symptoms of all gradations of severity are seen, and in cases of the acute edematous or acute interstitial form of the disease, as Elman [2] has pointed out, there may be no distinguishing characteristics and the disease may mimic any of the other causes of severe pain in the upper abdominal area such as cholecystitis, perforated ulcer, intestinal obstruction or coronary thrombosis. The diagnosis is established in these cases by the demonstration of an elevation of the serum amylase level.

Despite extensive experimental and clinical study, there remains wide divergence of opinion regarding the cause of acute pancreatitis. Most observers comment, however, on the frequent association of chronic disease of the gallbladder.

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1. Elman, R.: Surgical Aspects of Acute Pancreatitis with Special Reference to Its Frequency as Revealed by Serum Amylase: Max Bellin Lecture, J. A. M. A. **118**:1265-1268 (April 11) 1942.

2. Elman, R.: Nitroglycerine in the Treatment of Acute Pancreatitis, Am. J. Digest. Dis. **6**:474-475 (Sept.) 1939.

Of the various hypotheses which have been put forward to explain its origin, those which have gained widest acceptance are as follows: first, the reflux of bile into the pancreatic duct resulting from conversion of the common bile duct and the pancreatic ducts into a common channel by obstruction at the ampulla of Vater, by a calculus, from spasm of the sphincter of Oddi or from edema; second, obstruction of the pancreatic ducts by cysts or tumors or by epithelial metaplasia as described by Rich and Duff.[3] Other etiologic factors frequently mentioned are trauma, occlusion of the arterial or venous circulation of the pancreas by thrombosis or embolism and infection, lymph or blood borne, extending from adjacent organs or ascending the duct from the duodenum or the biliary tract.

Whatever the initiating cause, there seems to be substantial agreement that the subsequent changes which occur in the pancreas are related to the release of tryptic activity of the pancreatic proteolytic enzyme. The manner in which the inactive proenzyme trypsinogen becomes activated remains unsettled.

The pathologic changes which occur in acute pancreatitis vary, with the severity of the disease, from a diffuse edema of the organ and adjacent structures in the acute interstitial form to the most extensive destruction. In acute hemorrhagic necrosis the pancreas is converted into a tense, swollen, darkly mottled organ with areas of hemorrhage and necrosis of varying extent. There may be areas of suppuration, and in extreme cases the entire gland may be gangrenous. Areas of fat necrosis may be widespread over the parietal peritoneum and omentum or localized in the region of the foramen of Winslow. Varying amounts of consommé-like or hemorrhagic fluid are found in the free peritoneal cavity and in the lesser peritoneal sac.

It is to be expected that such extensive changes in the parenchyma of the pancreas will lead, in a certain percentage of nonfatal cases, to secondary sequelae after the acute process has subsided.

In the suppurative form of the disease, with extensive destruction of pancreatic tissue, localization of the process and abscess formation are likely to occur.

When extensive destruction of pancreatic tissue has occurred and when infection does not play a dominant role, conditions are set up which may give rise to the later development of so-called pseudocysts.

Pinkham[4] stated that pseudocysts may be formed within the substance of the pancreas as the result of degenerative changes affect-

3. Rich, A. R., and Duff, G. L.: Experimental and Pathological Studies on Pathogenesis of Acute Hemorrhagic Pancreatitis, Bull. Johns Hopkins Hosp. **58**:212-259 (March) 1936.

4. Pinkham, R. D.: Pancreatic Collections (Pseudocysts) Following Pancreatitis and Pancreatic Necrosis: Review and Analysis of Ten Cases, Surg., Gynec. & Obst. **80**:225-235 (March) 1945.

ing the interstitial tissues of the gland or adjacent to the pancreas when the corrosive action of the pancreatic ferments has led to encapsulated accumulations about the gland.

As in subdiaphragmatic abscess or any other inflammatory disturbance beneath the diaphragm, it would appear likely that an inflammatory process in the pancreas might occasionally result in the production of a pleural effusion in the adjacent thoracic cavity. This finding has been infrequently reported, but such cases have been described by Metheny [5] and Hultén.[6]

It has been suggested by Peterson and Cole [7] that acute edematous pancreatitis may lead to the later development of chronic fibrosing diffuse pancreatitis. They report 3 such cases.

In a series of 20 cases of acute pancreatitis, of which 12 were classified as acute edematous and 8 as acute hemorrhagic pancreatitis, all the aforementioned sequelae have been observed. Four cases are briefly reported:

CASE 1.—A woman of 68 was seized by a severe pain in the upper abdominal area associated with protracted nausea and vomiting. Examination showed generalized abdominal tenderness, most pronounced in the epigastrium and the left upper quadrant. The white blood cell count was 17,000 and the temperature 101.5 F. A clinical diagnosis of acute pancreatitis was made and later confirmed by the presence of a serum amylase level of 1,217 units. A roentgenogram of the abdomen showed gallstones. She was treated conservatively with supportive measures and sodium sulfadiazine and penicillin. Her course was stormy, but her general condition slowly improved. She continued to have a febrile course, however, and three weeks after admission a roentgenogram of the chest revealed pleural effusion on the left side. A subdiaphragmatic abscess on the left was suspected. A roentgenogram made after a barium enema and an intravenous pyelogram gave indication of a mass displacing the splenic flexure downward and distorting the upper pole of the left kidney. The left anterior subdiaphragmatic space was explored extraperitoneally, and nothing was found. The peritoneum was opened, and a large abscess of the pancreas was found. This was marsupialized, and almost 2 quarts (2,000 cc.) of thick yellow pus was evacuated. The patient's condition improved, but the pleural effusion increased and in the ensuing six weeks thoracentesis was done on the left side six times, with removal each time of from 700 to 1,500 cc. of hemorrhagic fluid. The cultures were sterile on all occasions. The fluid reaccumulated rapidly. The drainage from the abscess gradually diminished, her general condition improved and she was discharged nine weeks after admission with persistent pleural effusion. This slowly absorbed over a period of many months, and she finally made a full recovery and has recently been reported well.

5. Metheny, D.: Personal communication to the authors.

6. Hultén, O.: Beitrag zur Röntgendiagnose der abuted Pankreasaffektionen, Acta radiol. **9**:222-254, 1928.

7. Peterson, L. W., and Cole, W. H.: Chronic Sclerosing Pancreatitis Causing Complete Stenosis of Common Bile Duct, Arch. Surg. **51**:15-21 (July) 1945.

CASE 2.—A woman of 35 was admitted to the hospital three days after the onset of severe pain in the left upper abdominal area associated with persistent nausea and vomiting. Examination showed tenderness and spasm in the left upper quadrant, and just to the left of the umbilicus a firm tender mass the size of an egg was felt. She was operated on after a preoperative diagnosis of incarcerated internal hernia. Acute hemorrhagic pancreatitis was present. The mass was found to consist of matted adherent omentum. An ileostomy was done and a drain placed down to the pancreas. The patient's course was a stormy one, but she gradually improved until one month later, when a constant dull ache in the lumbar area developed. Several days later a mass was palpated high in the left upper quadrant. This rapidly increased in size. A series of roentgenograms of the upper gastrointestinal tract showed the stomach displaced downward and forward. At operation a large pancreatic pseudocyst was found containing cloudy blood-tinged fluid. This was marsupialized, and she made a complete recovery.

CASE 3.—A man aged 37 with a previous history of repeated attacks of severe midabdominal pain over a period of years was admitted to the hospital after the sudden onset of agonizing generalized abdominal pain associated with persistent nausea and vomiting. Examination showed generalized abdominal tenderness, most apparent in the left upper quadrant. The white blood cell count was 19,800, and the blood sugar content was 250 mg. per hundred cubic centimeters. A diagnosis of acute pancreatitis was made, and he was treated conservatively, recovered and was discharged, with advice to return for cholecystectomy.

Sixteen months later he returned with a complaint of nausea and vomiting and a constant dull ache in the upper part of the abdomen, with intermittent attacks of sharper pain. Two weeks later cholecystectomy was done. This showed chronic cholecystitis without stones.

Seven months later, two years after the initial attack, he returned, complaining of dull nonradiating pain in the left upper quadrant. Examination revealed a mass the size of a grapefruit in the left upper quadrant. A diagnosis of pseudocyst of the pancreas was made, and at operation this was confirmed. Approximately 2 quarts (2,000 cc.) of dark brown turbid fluid was evacuated. His convalescence was satisfactory until three weeks after the operation, when he had a severe hemorrhage from the draining sinus. In the next four days he had repeated severe hemorrhages, which were not controllable by packing of the cavity, and he died despite repeated transfusions. It was felt that the hemorrhage had resulted from erosion of a vessel by the corrosive action of pancreatic proteolytic enzyme.

CASE 4.—A man aged 32 was awakened on the morning of his admission to the hospital by a sudden severe pain in the right upper quadrant radiating across the midline to the left. This became progressively worse, and two hours later he vomited. Examination showed tenderness and spasm across the upper part of the abdomen, most pronounced on the right. He presented a peculiar grayish cyanosis. A diagnosis of acute pancreatitis was made, and he was operated on. The gallbladder was tense and thickened. The entire pancreas was swollen and somewhat thickened, and there was considerable edema of the adjacent posterior parietal peritoneum, and small droplets and rivulets of fluid could be seen beneath it. There were similar droplets of fluid beneath the pancreatic capsule. There was no fat necrosis. A diagnosis of acute edematous pancreatitis was made, and cholecystectomy was done.

He remained well until two years later, when he began having recurring attacks of distress and distention in the upper abdominal area, at times with nausea and vomiting. These attacks came about once or twice a month and continued on and off until two years later, when he was readmitted to the hospital with deep jaundice. A diagnosis of obstruction of the common duct due to pancreatitis was made, and he was operated on. The gallbladder was distended but contained no stones. The entire pancreas was thickened and fibrotic. These changes were most notable in the head. The common duct was opened, and a uterine sound was passed into the duodenum with the greatest difficulty. The instrument was gripped firmly in the intrapancreatic portion of the duct. Nothing larger could be passed. The duodenum was opened, and a catheter was fastened to the uterine sound and pulled up through the duct. A tapered vitallium® tube was then fastened to the catheter and pulled down through the duct. The gallbladder was then removed and the duct drained. He made an excellent recovery from the operation, and the jaundice cleared up and has not recurred, but he has continued to have periodic attacks of epigastric distress Ten months later he was readmitted to the hospital for study of his pancreatic secretions. The trypsin, diastase and lipase concentrations were all diminished but were well above the levels seen in cases of achylia pancreatica. It was felt, however, in view of the findings at operation and of these findings that this represented a case of chronic fibrosing pancreatitis resulting from a previous acute edematous pancreatitis.

SUMMARY

Significant surgical sequelae were observed five times in 4 cases among 20 patients suffering an attack of acute pancreatitis. These consisted of residual abscess formation, pseudocyst and chronic pancreatitis producing biliary obstruction.

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PATHOGENESIS OF POSTOPERATIVE PULMONARY ATELECTASIS

An Experimental Study

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ONE OF the most discouraging complications confronting a surgeon is the clinical entity commonly called postoperative pulmonary atelectasis. By postoperative atelectasis reference is made to that pulmonary complication which occurs within the first two or three postoperative days. This condition is accompanied with actual obstruction within the bronchial tree. The classic signs and symptoms of this condition include fever, leukocytosis, cough, purulent or tenacious sputum and an area in the lung of dulness to percussion and of diminished or absent breath sounds. The classic roentgenographic findings include diminution in lung volume, displacement of the mediastinum and narrowing of the overlying intercostal spaces.

Postoperative atelectasis should be differentiated from postoperative pneumonia, which may give rise to the same symptoms and even the same physical signs, before actual consolidation occurs. Clinically, a roentgenogram of the chest taken in expiration may differentiate the two conditions.[1] However, there are undoubtedly many combinations of these two conditions, such as pneumonitis behind an obstruction and an obstruction superimposed on a previously existing pneumonia.

It may be questioned whether the familiar postoperative complication should properly be called atelectasis. By strict definition, atelectasis means imperfect expansion, though clinically the term is commonly used to include collapsed pulmonary tissue. Certainly, collapsed pulmonary tissue is at least a part of so-called postoperative pulmonary atelectasis. If clinical atelectasis consists in collapsed pulmonary tissue alone, it is difficult to understand why it should give rise to fever, leukocytosis, cough and sputum. If simple collapse of pulmonary tissue causes such

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1. "In a true atelectasis, displacement toward the side of the lesion is present on expiration as well as on inspiration, while in consolidation this displacement is present on inspiration only" (Holmes, cited by King, D. S.: Surg., Gynec. & Obst. **56**:43, 1933.

symptoms, why do not such symptoms occur in patients with therapeutic pneumothorax? It would seem logical to conclude that the symptoms of postoperative atelectasis are due, rather, to pneumonitis, pneumonia or suppuration which exists peripheral to the point of obstruction. On the other hand, if such an infectious process exists beyond the obstruction, it is difficult to understand why the process is so rapidly and dramatically relieved by removal of the obstructing factor.

Lamar and Meltzer [2] concluded that the occlusion of air passages is an aid in the production of experimental lobar pneumonia, and Coryllos and Birnbaum [3] even expressed the opinion that the obstruction of air passages is an essential factor in the production of lobar pneumonia. Coryllos and Birnbaum [4] also stated that atelectasis might predispose to an infectious process in the parenchyma of the lung by localization of organisms from the circulating blood.

It is felt that obstruction of the air passages may predispose the lung to infection but that atelectasis per se does not do so. The concept of the misuse of the term atelectasis was obtained from Dr. J. Burns Amberson,[5] who wrote:

> I think the diagnosis of atelectasis in the specific sense is usually erroneous, because as a rule there is little or no collapse of the alveoli, but, rather, an accumulation of edema fluid, which is often soon followed by pneumonia. . . . The important concept, which is fundamental, is that collapse of the alveoli in itself does not predispose to pneumonia, whereas edema always does.

Gunn [6] also stated the belief that edema fluid predisposes to pneumonia.

Coryllos [7] pointed out the close relation of the postoperative pulmonary complications usually described as postoperative bronchitis, atelectasis and pneumonia, and he expressed the opinion that generally these conditions follow one another in the order named, without clear-cut, distinctive signs and symptoms. Undoubtedly, there are combinations of pulmonary complications. The purpose of the present study was to determine the part played by obstruction.

In the presence of bronchitis existing prior to obstruction of a bronchus, one would expect the occurrence of secretions and organisms

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2. Lamar, R. V., and Meltzer, S. J.: Experimental Pneumonia by Intrabronchial Insufflation, J. Exper. Med. **15**:133, 1912.

3. Coryllos, P. N., and Birnbaum, G. L.: Lobar Pneumonia: Considered as Pneumococcic Lobar Atelectasis of the Lung; Bronchoscopic Investigation, Arch. Surg. **18**:190 (Jan., pt. 2) 1929.

4. Coryllos, P. N., and Birnbaum, G. L.: Obstructive Massive Atelectasis of the Lung, Arch. Surg. **16**:501 (Feb.) 1928.

5. Amberson, J. B.: Personal communication to the author.

6. Gunn, F. D.: Pathogenesis of Pneumonia, M. Clin. North America **32**:9 1938.

7. Coryllos, P. N.: Postoperative Pulmonary Complications and Bronchial Obstruction, Surg., Gynec. & Obst. **50**:795, 1930.

beyond the point of obstruction, and it would seem probable that such a condition is favorable to the development of an infection within the involved lung. However, bronchial obstruction alone, causing a dry atelectasis, is presumably not harmful in itself. Adams and Livingstone,[8] as well as Allen,[9] showed that pulmonary suppuration is not associated with bronchial obstruction alone as produced experimentally in dogs.

What proof is there that bronchial obstruction will aid in the production of pulmonary infection and in increasing its severity? The answer to this question varies somewhat with the organisms involved. Allen [9]

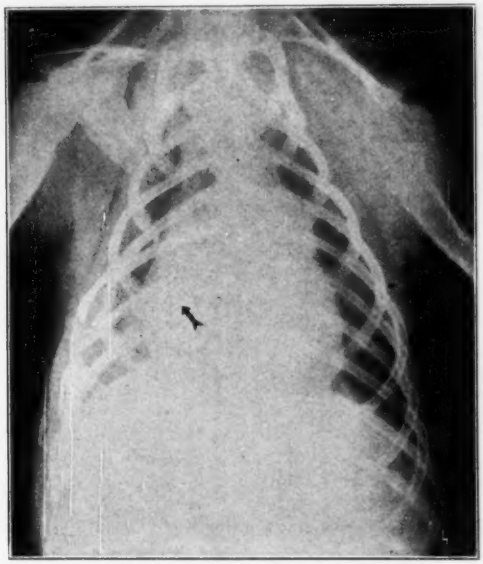

Fig. 1.—Roentgenogram of the chest of a dog in expiration, two days after ligation of the main bronchus to the middle lobe of the right lung.

showed that obstruction of the bronchus, producing a collapse of pulmonary tissue, plays a prominent part in the formation of experimental aspiratory anaerobic abscesses of the lung. On the other hand, Adams and Vorwald [10] showed that obstruction of the bronchus, with the

8. Adams, W. E., and Livingstone, H. M.: Obstructive Pulmonary Atelectasis, Arch. Surg. **23**:500 (Sept.) 1931.

9. Allen, D. S.: Etiology of Abscess of the Lung, Arch. Surg. **16**:179 (Jan., pt. 2) 1928.

10. Adams, W. E., and Vorwald, A. J.: The Treatment of Pulmonary Tuberculosis by Bronchial Occlusion, J. Thoracic Surg. **3**:633, 1934.

resulting collapse of pulmonary tissue, actually enhances the healing of tuberculous (aerobic) infections. It would seem that ventilation aids the growth of aerobic organisms and lack of ventilation favors the growth of anaerobic organisms.[11]

Whether or not atelectatic pulmonary tissue is a site of predilection for certain organisms circulating in the blood stream has been considered. In a small series of dogs, Adams [12] found that septic and aseptic emboli containing lead pellets were twice as likely to lodge in atelectatic as in inflated pulmonary tissue, whereas tuberculous (aerobic) emboli were much less likely to lodge in atelectatic than in inflated pulmonary tissue.

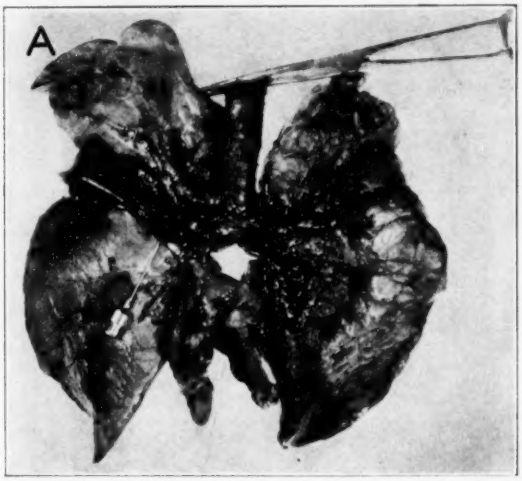

Fig. 2 (group 1).—*A*, ligation of the main bronchus of the middle lobe of the right lung two days before the dog was killed. The needle points to the site of ligature.

PROCEDURE

The following experiment on dogs was an attempt to throw light on the part played by bronchial obstruction in postoperative pulmonary complications. Obstruction of the bronchus was produced by ligation of the bronchus with a soft, strong cotton string. Proof of the complete occlusion was evident in the

11. Coryllos, P. N.: How Do Rest and Collapse Treatment Cure Pulmonary Tuberculosis? J. A. M. A. **100**:480 (Feb. 18) 1933.

12. Adams, W. E.: Rapid Healing of Embolic Lung Abscess in Atelectatic Lobes, Proc. Soc. Exper. Biol. & Med. **29**:539, 1931.

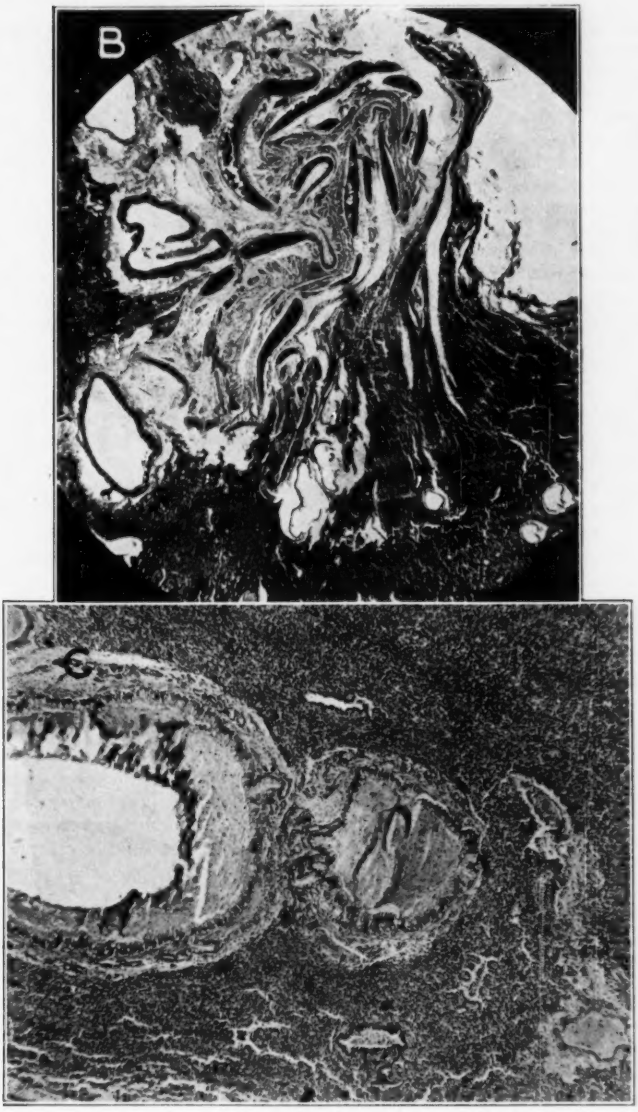

Fig. 2.—*B*, section taken at the site of ligature of bronchus, showing inflammatory reaction and area of necrosis due to ligature with atelectasis. *C*, ligation of the bronchus, with accumulation of mucus within the bronchioles and atelectasis.

inability to pass injected water through the obstructed bronchus. The obstruction was observed by a microscopic section taken at the level of the ligature. The bronchus of the middle lobe of the right lung was selected because of its anatomic accessibility. Routine anesthesia induced by intravenous injection of 5 per cent pentobarbital sodium U. S. P. (2.7 cc. per pound, 6 cc. per kilogram, of body weight) was used. The chest was entered through the right fifth interspace. An artificial respirator, constructed from an electric fan motor and connected to an air pressure system, supplied alternating pressure through an intratracheal tube. This device gave satisfactory aeration.

A virulent organism to which dogs were susceptible was used. A type I pneumococcus, designated as A5/8D 102R, was obtained from Dr. O. H. Robertson, and its virulence was maintained according to his specifications.[13] This pneumococcus grew better on the surface than it did in the depths of stab inoculations of rabbit serum agar, although it did grow in the depths also. Septicemia was produced in 2 normal dogs by intravenous injections of 1 and 3 cc., respectively, of broth culture. Pneumococci were recovered from the heart blood or from the splenic pulp at autopsy. No gross evidence of involvement of the lungs was noted.

The introduction of sterile gastric mucin with the pneumococcus was considered, since mucin has been known to increase the likelihood of producing experimental pneumonia,[14] but this substance was not used, as it was deemed wiser to avoid the introduction of more factors than was necessary.

Four groups of dogs were used, in order that the factor of obstruction might be evaluated.

Group 1.—The main bronchus of the middle lobe of the right lung was ligated.

Group 2.—The pneumococcus culture, 0.25 cc., was injected into the bronchus of the right middle lobe by means of a curved hypodermic needle passed through the wall of the bronchus in a manner causing the injected medium to pass toward the periphery of the lobe.

Group 3.—The bronchus was both ligated and injections made into it in a manner identical to that described in groups 1 and 2, the injections being given peripheral to the ligature.

Group 4.—The dogs were subjected to ligation of the bronchus of the middle lobe of the right lung. This procedure was followed, either immediately or several days later, by the intravenous injection of the pneumococcus culture. The lower lobe of the right lung was traumatized in order that the factor of manipulative trauma might be evaluated. No evidence of any effect of such trauma was noted.

These dogs were followed clinically and killed on the first, second or third day after treatment, or occasionally later. On a number of dogs preoperative

13. The pneumococci were kept in a stock culture of heart infusion broth (Difco) to which was added 2 per cent whole rabbit blood. In growing it for use, two or three loopfuls were planted into serum-dextrose broth (heart infusion broth plus 2 per cent serum, either rabbit or horse, and 0.5 per cent dextrose). To keep up its virulence, the organism was passed through a rabbit every six weeks. (Dr. O. H. Robertson, in a personal communication to the author.)

14. Nungester, W. J., and Jourdonais, L. F.: Mucin as an Aid in the Experimental Production of Lobar Pneumonia, J. Infect. Dis. **59**:258, 1936.

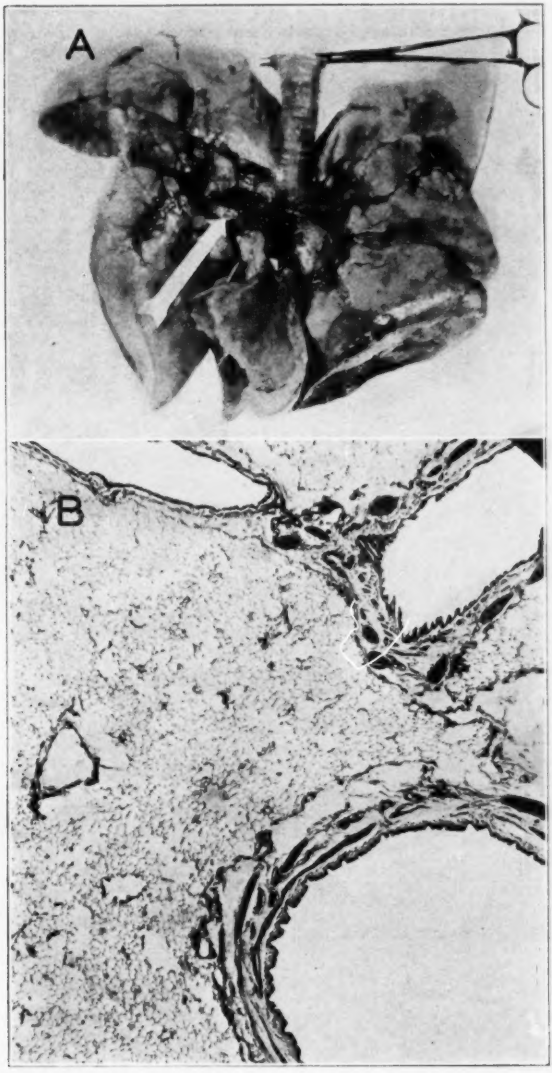

Fig. 3.—*A* (dog from group 2), injection of pneumococcus into the main bronchus of the middle lobe of the right lung one day before the dog was killed. The arrow points to the site of injection. *B*, lung of a normal dog.

and postoperative rectal temperatures and white blood cell counts were determined. In a number of dogs preoperative and postoperative roentgenograms were taken of the chest.

The temperature and white blood cell values were recorded. Rectal temperatures in all four groups showed no significant changes except for the 2

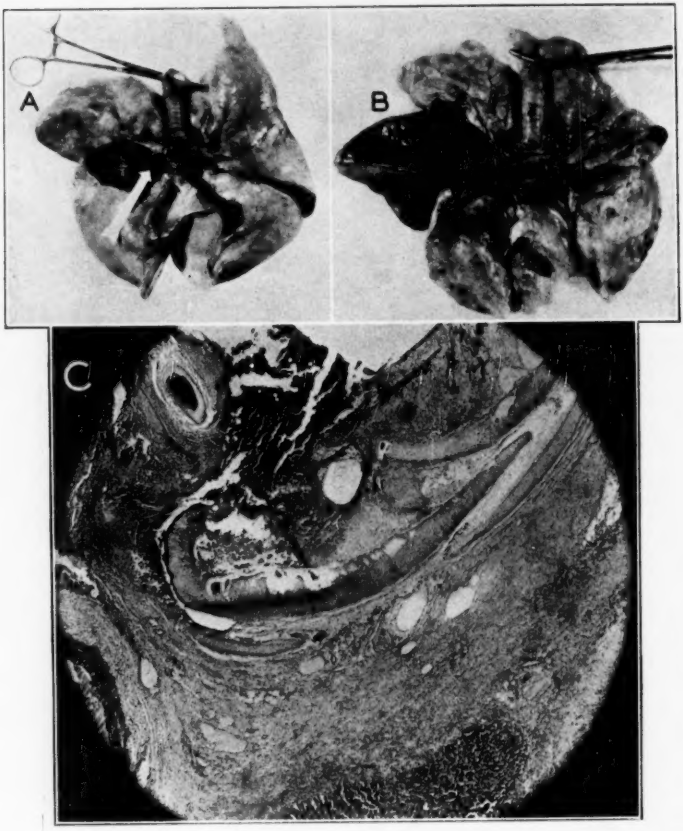

Fig. 4 (group 3).—*A*, ligation and injection of the main bronchus of the middle lobe of the right lung one day before the animal was killed. The arrow points to the site of the ligature. *B*, ligation and injection of the main bronchus of the middle lobe of the right lung three days before the dog was killed. *C*, ligation and injection of bronchus, showing purulent bronchitis and peribronchitis, with necrosis of the bronchial wall, and atelectasis with scanty leukocytic infiltration.

acutely ill, dying dogs, whose temperatures were increased over 3 degrees (F.), and a third dog, whose temperature dropped 3 degrees (F.) just before death.

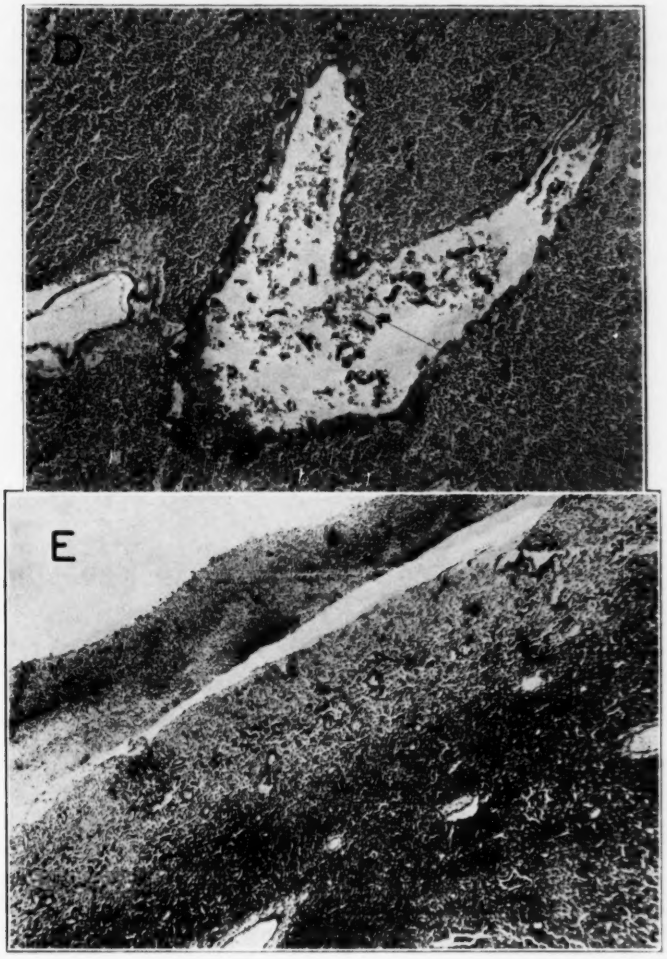

Fig. 4.—*D*, ligation and injection of bronchus and showing bronchiolitis, with desquamation of the bronchiolar epithelium into the lumen, pneumonia. *E*, ligation and injection of bronchus, with fibrinocellular exudate on the pleural surface, atelectasis and pneumonia.

The thoracotomies seemed to have no appreciable effect on the temperature reactions of these dogs. The white cell counts showed changes in either direction after various procedures. In most instances there was some leukocytosis following thoracotomy with or without ligation of a bronchus. After the intravenous injection of the pneumococci culture leukocytosis was usually noted. Two of the acutely ill dogs had pronounced leukopenia. No attempt was made to determine the heart rate in these dogs, and none showed evidence of coughing. It would seem that the dog does not lend itself to a clinical comparison with man, at least not so far as temperature, white blood cell counts and cough are concerned.

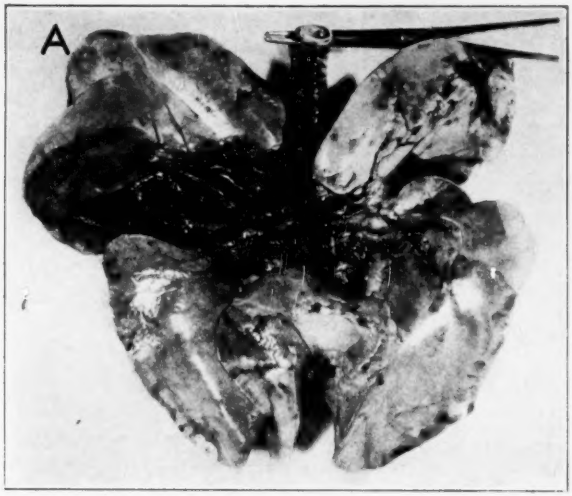

Fig. 5 (group 4).—*A*, ligation of bronchus eleven days, and intravenous injection three days, before the dog was killed. Note the small nodular elevations of the pleural surface.

RESULTS

A number of roentgenograms were taken of the dogs' chests, and variations were noted in the four groups. Atelectasis was evident in group 1. Group 2 showed no demonstrable changes. A number of dogs in group 3 showed evidence of consolidation. The picture in group 4 showed no definite changes toward consolidation and appeared to be similar to the atelectatic picture seen in the dogs of group 1.

The results in each group were followed by tabulating the type and weight of each dog, the postoperative course, the postoperative day on which the dog was killed and the autopsy observations, gross and microscopic, of the lobes that were obstructed or had received

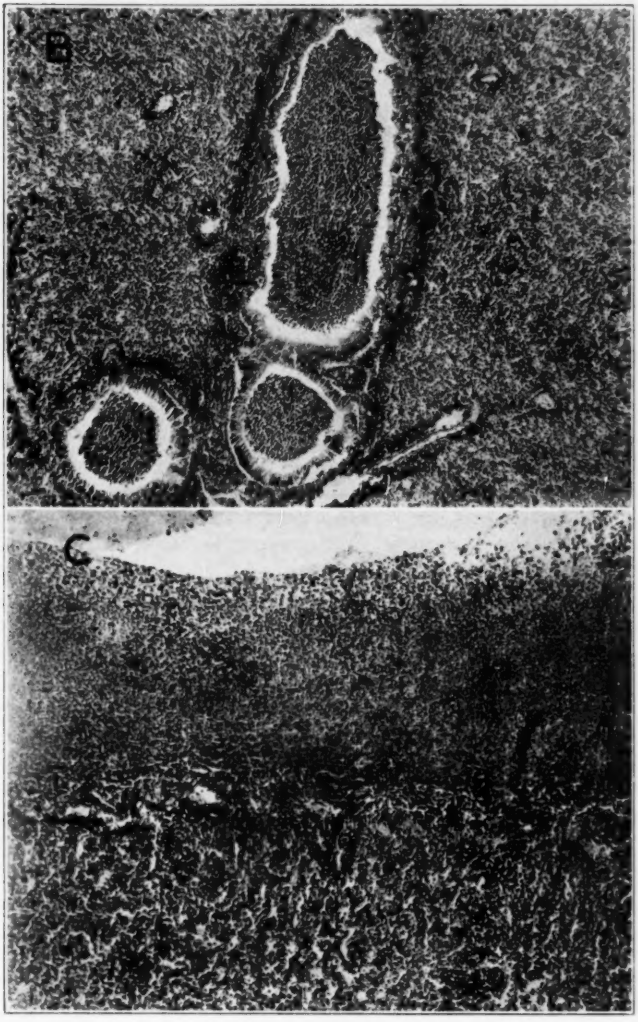

Fig. 5.—Effect of ligation of the bronchus and intravenous injection showing (*B*) bronchiolitis with purulent exudate, peribronchiolitis and bronchopneumonia with leukocytic exudate in the alveoli, and (*C*) fibrinous pleuritis, with red blood cells and neutrophils within the fibrin, and bronchopneumonia in the atelectatic lung.

injections and of those that were not obstructed or had not received injections.

GROUP 1.—The ligation of the main bronchus to the middle lobe of the right lung alone did not affect the immediate health of the dogs, and the pathologic changes were simply those of atelectasis. The accumulation of mucus within the lumen of the bronchi and bronchioli was noted beyond the point of obstruction. Acute inflammatory exudate in the bronchial lumen and pleuritis were not in only 1 of the 6 dogs.

GROUP 2.—The intrabronchial injection of pneumococci produced no appreciable signs of disease, and in only 1 of 6 dogs was pneumonia produced. Why the pneumonia occurred in this dog, and not in the others, is a matter of conjecture. The presence of bronchial obstruction was not demonstrated. Perhaps the resistance of the particular host was not great enough to overcome the virulence of the pneumococci. No pleuritis was noted in any of the lobes.

GROUP 3.—The dogs in which the bronchus of the middle lobe of the right lung was both ligated and given direct injections of pneumococci all showed leukocytic infiltration of the parenchyma and pleuritis. Two of the lobes not having bronchial obstruction or pneumococcic injection showed areas of focal leukocytic infiltration, but otherwise seemed normal.

GROUP 4.—The bronchus of the middle lobe of the right lung was ligated and immediately or subsequently thereafter an intravenous injection of the pneumococcus culture was given. Bronchopneumonia developed in the collapsed lobe in 1 of these 7 dogs. Purulent exudate in the bronchi of the collapsed lobes was noted in all 7 dogs of this group. Of the nonobstructed lobes of these 7 dogs there was an area of slight focal leukocytic infiltration in 1 and an area of bronchopneumonia in another. Four of the 7 collapsed lobes showed definite pleuritis, while no involvement of the pleura could be demonstrated in the aerated pulmonary tissue except for an interlobar empyema on the surface of a collapsed portion of a lobe adherent to a ligated lobe.

COMMENT

These observations more than suggest that the obstructed lobes have a greatly increased susceptibility to the pneumococcus when the organism is introduced directly into the bronchial lumen of the ligated lobe. This susceptibility would seem to be due to the trapping of the organisms within the obstructed lobe. The presence of accumulated mucus may enhance the growth of the trapped organisms,[14] and the lack of drainage prevents the expulsion of the organisms. Poor ventilation does not seem to be the responsible factor, since this organism

grows better in the presence than in the absence of air. It would seem likely that the uniform pleural involvement was secondary to the enclosed pneumonitis.

The intravenous injection of pneumococci in mice is known to produce pneumonia,[15] but the results have rarely been obtained in other experimental animals. Kempf and Nungester [16] produced pneumonia in rats after using intrabronchial innoculation of sterile mucin, followed by the intravenous injection of pneumococci. They concluded that "the presence of mucous material in the lung or some comparable condition is a necessary predisposing factor for the localization of the pneumococci." [17]

In the fourth group of dogs the susceptibility to infection of the ligated lobe from the blood stream seemed definitely greater than that of the aerated lobes. This ratio is further increased by the fact that there are six aerated lobes to each obstructed lobe. The pleura was involved more often than was the lung parenchyma. In this group of dogs there was no trapping of organisms within a "bottled-up" lobe, as was the case in the third group. Is it not possible that the presence of mucus, accumulated behind the obstruction, is the initiating cause of the increased susceptibility to the pneumococcus? [8] Such a postulate would seem likely in the development of infections in the bronchial tree and alveoli, as well as in the pleura, which presumably becomes involved by extension from the lung parenchyma. Trauma to the surface of the lobe during the operative procedure is apparently not the factor producing the pleuritis, since trauma of like or greater severity was applied to the adjacent lower lobe of the right lung without producing pleuritis.

The lack of drainage of both mucus and organisms is doubtless an aid to the continuation of infection behind an obstruction, whether the organism is introduced intrabronchially or intravenously.[17]

Although the evidence may be presumptive, it seems that the most likely cause of the increased susceptibility to pneumococcic infection of the ligated lobes was the increased mucus content of those lobes. Such a postulate is in accord with the work of Kempf and Nungester [16] and others,[18] who noted the greatly increased susceptibility of rats to pneu-

15. Rake, G.: Pathogenesis of Pneumococcus Infection in Mice, J. Exper. Med. **63**:191, 1936.

16. Kempf, A. H., and Nungester, W. J.: Production of Pneumonia in Rats by Intravenous Injection of Pneumococci, Proc. Soc. Exper. Biol. & Med. **43**:627, 1940.

17. Coryllos and Birnbaum.[8] Nungester, W. J., and Klepser, R. G.: A Possible Mechanism of Lowered Resistance to Pneumonia, J. Infect. Dis. **63**:94, 1938.

18. Robertson, O. H.; Coggeshall, L. T., and Terrell, E. E.: Experimental Pneumococcus Lobar Pneumonia in the Dog, J. Clin. Investigation **12**:433, 1933.

mococci introduced intrabronchially and intravenously when sterile gastric mucin was instilled into the bronchial tree.

This experiment has not included any therapeutic measures, but the evidence in favor of the assumption that excess intrabronchial mucus is a detrimental factor is further substantiated by the clinical relief obtained by the removal of bronchial plugs and bronchial mucus in cases of postoperative pulmonary atelectasis.

In a further study of the importance of mucous contents in the bronchial tree of the atelectatic lung, it would be interesting to compare the respective amounts of mucus in pulmonary lobes collapsed by compression, as in pneumothorax, in lobes collapsed by bronchial obstruction. Would the nonobstructed lobes reveal less mucus, owing to partial, if not complete, drainage? If such were to prove the case, would there be any demonstrable difference in the susceptibility of the two types of collapsed lobes to pneumococcic infection?

Mucus may not be the only factor producing this enhancement of effective virulence of certain organisms. The fluids produced by such factors as congestive heart failure, trauma and irritants may well play a part in the increased susceptibility of pulmonary tissue to infectious organisms. However, mucus is ever present and would seem to be the substance most usually causing this increased susceptibility in persons previously possessing normally functioning lungs.

It would seem that the condition commonly referred to as postoperative pulmonary atelectasis is not merely bronchial obstruction with subsequent atelectasis. In most instances postoperative pulmonary atelectasis is initiated by bronchial obstruction, which, in turn, produces a certain amount of alveolar collapse but also leads to the accumulation of mucus. The mucus, in turn, may aid in the localization and prevent the dispersion of certain organisms, including the pneumococcus. The infection thus produced is presumably the cause of such symptoms as leukocytosis and fever, rather than the obstruction and alveolar collapse per se. Removal of the obstruction not only permits the inflation of alveoli but allows the evacuation of mucus, and it is probably the riddance of the accumulated mucus and the associated organisms which permits the patient to recover. It is logical that the earlier the mucus is evacuated the more rapid will be the recovery. Such a belief is substantiated by clinical therapeutic results.[19]

SUMMARY AND CONCLUSIONS

Experiments were carried out on four groups of dogs:

In the first group the ligation of the main bronchus of the middle lobe of the right lung produced atelectasis of this lobe and the accumu-

19. Schmidt, H. W.; Mousler, L. H., and Harrington, S. W.: Postoperative Atelectasis, J. A. M. A. **120**:895 (Nov. 21) 1942.

lation of mucus within the ligated bronchial tree. No appreciable detrimental effect on an otherwise normal dog was produced by this procedure. In the second group, a type I pneumococcus was injected into the lumen of the main bronchus of the middle lobe of the right lung, and in 5 of the 6 dogs no infection of the lobe was noted. In the third group, ligation of the main bronchus of the middle lobe of the right lung was accompanied with the intrabronchial injection of pneumococci peripheral to the point of obstruction. In all 7 dogs the collapsed lobes showed evidence of bronchitis, leukocytic infiltration of the parenchyma and pleuritis. In the fourth group, ligation of the bronchus of the middle lobe of the right lung was followed by the intravenous injection of pneumococci. The obstructed lobes were more susceptible to infection than were the nonobstructed lobes. Purulent bronchitis was the common finding, and 4 of the 7 dogs revealed pleuritis of the ligated lobes.

The following conclusions were formed as a result of this study: Obstructive atelectasis and pulmonary infection in dogs do not seem to give rise to the classic symptoms found in man, such as elevations of temperature, leukocytosis and cough.

The pulmonary changes in dogs have produced roentgenograms analogous to those found in man.

The factor in the production of infection in postoperative pulmonary atelectasis seems to be the accumulation of mucus in the bronchial tree, rather than the collapse of pulmonary tissue.

Black Mountain, N. C. (P. O. Box. 913).

NONSUTURE METHOD FOR VASCULAR ANASTOMOSIS UTILIZING THE MURPHY BUTTON PRINCIPLE

HARRY H. LeVEEN, M.D.
NEW YORK

IN 1900 Payr [1] introduced a nonsuture method of anastomosis employing a magnesium tube. One cut end of the vessel was drawn through the tube and everted over its edge, the intimal surface thus being brought to the outside. The vessel was held in this position by a ligature. The other opened cut end of the vessel was then drawn over the tube and secured again by ligature. The forceful electrochemical activity brought about by the presence of magnesium in tissues produced widespread necrosis.[2] Such anastomoses were, therefore, doomed to failure. More recently, Lord, Blakemore and Stefko [3] have introduced a vitallium® tube which is used in the same way as the original Payr tube but has the advantage of being made of a metal which does not produce tissue reaction. By use of this tube, these authors have advanced the scope of vascular surgery.[4] Their method, however, has technical disadvantages, making its use much more difficult than one might suppose. It is not well adaptable to deep abdominal surgery because of difficulty in placing ties and slippage of knots incident to tying on a rigid metal structure.

A method of anastomosis is herein suggested employing the nonsuture technic and utilizing a rigid tube [5] of teflon® plastic, which sub-

From the Department of Surgery, New York University School of Medicine.

1. Payr, E.: Beiträge zur Technik der Blutgefäss, und Nervennaht nebst Mittheilungen über Verwendung eines resorbirbaren Metales in der Chirurgie, Arch. f. klin. Chir. **62**:67, 1900.

2. Venable, C. S., and Stuck, W. G.: A General Consideration of Metals for Buried Appliances in Surgery, Surg., Gynec. & Obst. **76**:297, 1943.

3. Blakemore, A. H.; Lord, J. W., and Stefko, P. L.: Restoration of Blood Flow in Damaged Vessels, Ann. Surg. **117**:481, 1943.

4. Blakemore, A. H., and Lord, J. W.: The Technique of Using Vitallium Tubes in Establishing Portal Caval Shunts for Portal Hypertension, Ann. Surg. **122**:476, 1945.

5. The entire device is available from the Zack Manufacturing Company, Berwyn, Ill.

stance has been found nonabsorbable and free of tissue reaction.[6] The original work was done with nylon,[5] but this substance produced some tissue reaction which did not occur with teflon.[5] The principle involved is somewhat similar to the Murphy button principle and allows for satisfactory anastomosis with maximum ease and rapidity.

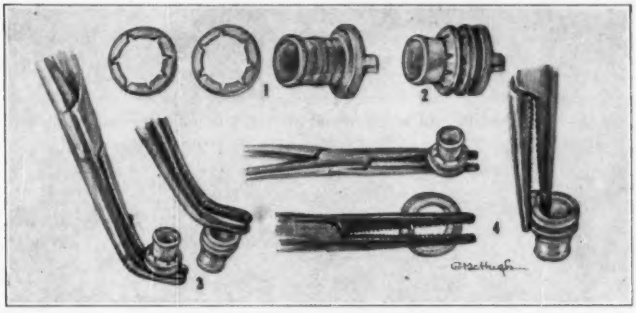

Fig. 1.—Component parts of the device for anastomosis and suggested methods of holding the tube to secure maximum stability.

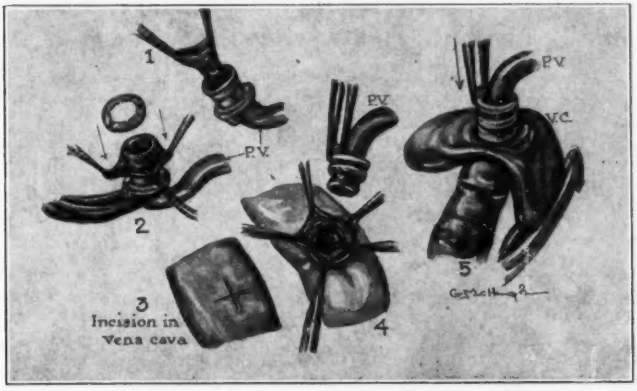

Fig. 2.—The various steps in the portal caval shunt operation. After ligation and division of the portal vein (*P.V.*) the cut end (occluded by a bulldog clamp) is pulled through an anastomosis tube and secured (*1* and *2*). A cruciate incision is made in the occluded vena cava (*3*). The cut edges of the incision are drawn through the anastomosis ring (*4*). With a finger placed beneath the vena cava for support, the prepared portal vein is pushed through the ring (*4*) to complete the anastomosis (*5*).

6. LeVeen, H. H., and Barberio, J. R.: Tissue Reaction to Plastics Used in Surgery with Special Reference to Teflon, Ann. Surg. **129**:74, 1949.

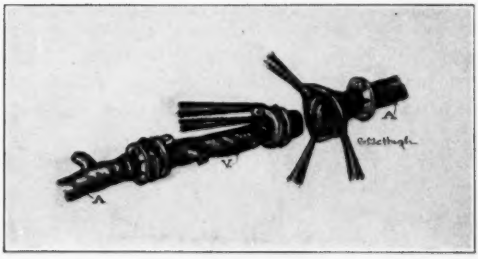

Fig. 3.—A method by which end to end arterial anastomosis could be performed.

Fig. 4.—The venograms were made by injecting a small venule in the mesentery of the small bowel with 70 per cent diodrast®. The free flow into the vena cava is demonstrated. Both venograms were taken six weeks after portal caval shunt. *A*, dog 281; *B*, dog 342.

DESCRIPTION OF METHOD

The apparatus for anastomosis is illustrated in figure 1. It consists of two scalloped nylon® rings and a teflon® tube. The rings slip over the free ends of the tube as illustrated and are secured by the spring action of their scalloped edges as well as by the small ridges on the outer surface of the tube. A small projection from the basal rim serves as a handle for holding and manipulating the tube. The entire device is sterilized by boiling.

End to Side Anastomosis.—This procedure is illustrated by an Eck fistula operation (portal caval shunt) performed on dogs.

After adequate exposure has been obtained (a Z, T or L-shaped incision is used) the inferior vena cava is freed for a distance of 2 inches (5.08 cm.) in the region where the anastomosis is to be performed. This is usually about an inch

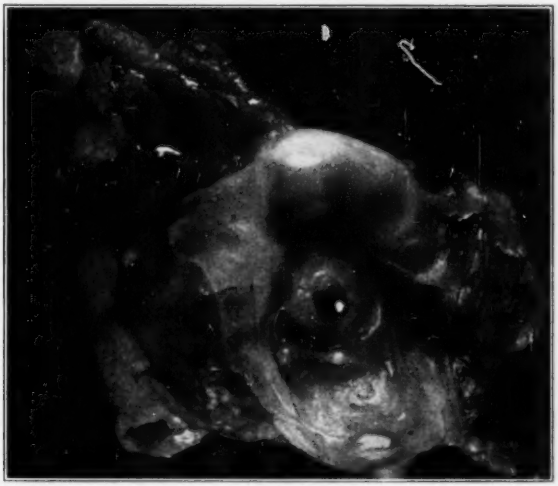

Fig. 5.—Photograph of a gross specimen removed at autopsy. The vena cava has been opened to show the patent portal anastomosis. This specimen was removed six weeks postoperatively. The small amount of perivascular fibrosis seen here does not occur with teflon® tubes.

(2.54 cm.) above the entrance of renal veins. After the vena cava has been freed, it should be possible to introduce the index finger beneath it with ease.

The portal vein is then isolated to the point where it is formed by the junction of the superior and inferior mesenteric veins. A Dieffenbach clamp is placed on it at this juncture. Ligation and division of the portal vein is performed just below the splenic vein. If it is desired to shunt the splenic circulation as well, ligation and division should be performed immediately beneath the bifurcation of the portal vein into the right and left hepatic veins. The vena cava is then clamped above and below the point of anastomosis with nontraumatic clamps.

The exact details of this anastomosis are illustrated in figure 2. Extreme care should be exercised in the selection of the size of the tube. It is important to prevent excessive tension and traction on the vein lest the thin-walled vessel be

torn or frayed. The diameter of the tube should be 1 mm. smaller than the diameter of the portal vein. If one is to err, it is better to err on the side of smallness. The opened end of the prepared portal vein should be moistened with heparin in isotonic sodium chloride solution. It would also seem advisable to administer heparin postoperatively.[7]

End to End Anastomosis.—End to end anastomosis may be satisfactorily performed as is illustrated in figure 3. This type of anastomosis is most suited to arterial work, with which the author has had no experience. The use of any nonsuture device for arterial anastomosis seems ill advised for reasons to be mentioned later.

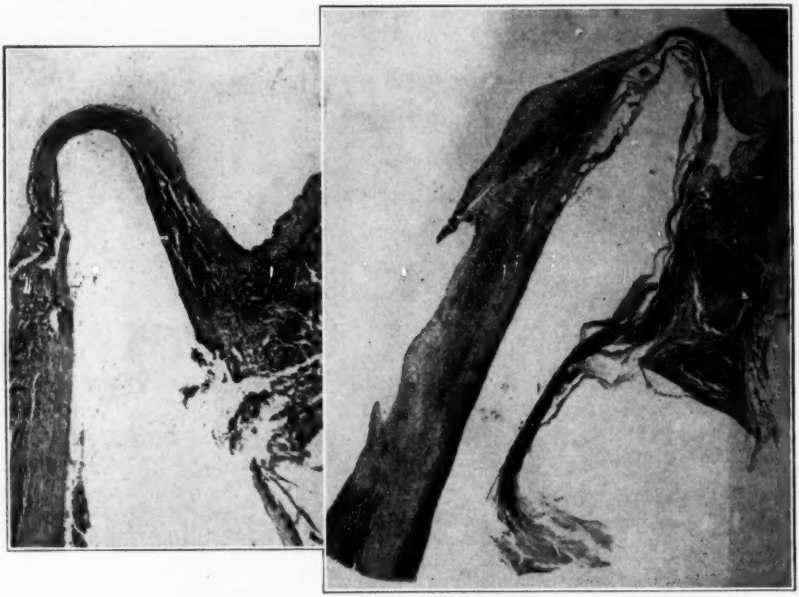

Fig. 6.—Photomicrographs of healed anastomosis showing absence of foreign body reaction. Healing is complete, and the exact area of joining is no longer discernible.

RESULTS OF SURGICAL PROCEDURES ON DOGS

An Eck fistula was performed in 20 dogs. Of 8 dogs which died in the first forty-eight hours, in none was the death attributable to blockage of the portal vein. Almost all the dogs in this group were found to have distemper, with pneumonic consolidation. Among the 12 that survived more than forty-eight hours, death from portal thrombosis occurred after three days in only 1 dog. The remainder of the dogs

7. Murray, G.: Heparin in Surgical Treatment of Blood Vessels, Arch. Surg. 40:307 (Feb.) 1940.

were killed after periods ranging from ten days to four months after operation for postmortem examination. Among the last 10 dogs operated on there was only 1 death, which was caused by distemper. In almost all the dogs the intimal surface of the portal vein was moistened with heparin and isotonic sodium chloride solution. Some dogs were given a small injection of heparin postoperatively.

The patency of the anastomoses was adequately illustrated by venograms taken at the time of autopsy (fig. 4). Autopsies on all dogs surviving more than forty-eight hours (with the exception of the one in which thrombosis occurred at the site of anastomosis) showed well healed areas of intima to intima anastomosis (fig. 5). There was no evidence of tissue reaction except for a few adhesions at the operative site, which are explainable on the basis of trauma at the time of operation or possibly of talc from the rubber glove (fig. 6).

COMMENT AND CONCLUSIONS

The need for speed in some forms of vascular surgery is imperative. In portal caval shunt operations, occlusion of the portal vein for a period of more than thirty minutes is hazardous since it may produce fatal shock in normal dogs or human beings. The method of anastomosis which has been described is rapid and simple. The results of anastomosis compare favorably with other methods.

It has some limitations, however, common to methods employing the use of rigid tubes.[8] A percentage of large arteries undergo necrosis at the site of contact of the artery with the rigid tube and rupture. In the case of the aorta, rupture may be produced by any circular structure placed around it and contacting the adventitia.[9] For this reason it probably would be best not to attempt the anastomosis of large arteries by the nonsuture method, although its use with small and medium-sized vessels would seem justified. For arterial anastomosis, a different type of locking ring must be used to avoid uniform circumferential pressure on the adventitia and to allow enough clearance for the relatively thicker arterial wall.

As yet no human being has been treated by this method although its extreme simplicity and favorable results as compared with other methods would warrant its use.

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8. Murray, G., and Janes, J. N.: The Healing of Arteries and the Relationship to Hemorrhage, Surgery **18**:624, 1945.

9. Pearse, H. E.: Experimental Studies on the Gradual Occlusion of Large Arteries, Ann. Surg. **112**:923, 1940.

SUMMARY

A simplified method for performance of vascular anastomosis has been described. The method adapts the Murphy button principle to small plastic tubes.

The results of the method which was employed in dogs are discussed and analyzed.

Mr. William Zack, of the Zack Manufacturing Company, designed the anastomosis tubes.

LYMPHOSARCOMA OF THE DUODENUM

Report of a Case; Review of the Literature

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AND

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THE LITERATURE on lymphosarcoma of the duodenum, the jejunum and the intestinal tract in general is replete with numerous comprehensive surveys, in addition to reports of small groups of cases emphasizing various diagnostic problems and findings.[1] Much of the material is valueless for statistical purposes because of the chaotic state of the nomenclature, inadequate reporting and poor follow-up observations on the patients.

From the Kennedy Veterans Hospital.

Presented at the Southern Surgeons Club, Atlanta, Ga., May 19 to 20, 1947.

*Dr. Copeland was formerly Chief of Surgery at the Kennedy Veterans Hospital at Memphis.

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1. (*a*) Assman, H.: Die klinische Roentgendiagnostik der inneren Ekrankungen, Leipzig, F. C. W. Vogel, 1930. (*b*) Beer, E.: Lymphosarcoma of the Small Intestine, S. Clin. North America **5**:93-96, 1925. (*c*) Berman, H., and Mainella, F.: Lymphosarcoma of Small Intestine, Am. J. Surg. **70**:121-125, 1945. (*d*) Benjamin, E. L., and Christopher, F.: Primary Lymphosarcoma of the Small Intestine, Am. J. Clin. Path. **10**:408-413, 1940. (*e*) Borden, D. L., and Taylor, F. D.: Primary Lymphosarcoma of Small Intestine: Case, Mil. Surgeon **92**: 255-257, 1943. (*f*) Callender, G. R.: Tumors and Tumor-Like Conditions of the Lymphocyte, the Myelocyte, the Erythrocyte and the Reticulum Cell, Am. J. Path. **10**:443-466, 1934. (*g*) Cave, H. W.: Tumors of the Small Intestine, Ann. Surg. **96**:269-285, 1932. (*h*) Charache, H.: Primary Lymphosarcoma of Intestine, Am. J. Surg. **27**:171-173, 1935; (*i*) Primary Lymphosarcoma in Boy of Seven: Follow-Up of Nine Years, ibid. **59**:601, 1943. (*j*) Cheever, D.: Clinical Aspects and Treatment of Primary Lymphosarcoma of the Stomach and Intestines, Ann. Surg. **96**:911-923, 1932. (*k*) Chont, L. K.: Sarcomas of Small Intestine and Reference to Their Radiosensitivity, Radiology **36**:86-97, 1941. (*l*) Cohn, S.; Landy, J. A., and Richter, M.: Tumors of Small Intestine, Arch. Surg. **39**: 647-666 (Oct.) 1939. (*m*) Crowther, C.: Studio dei sarcome primitive dell'intestino tenue con contributio di tre casi originale, Clin. chir., Milano **21**:2107-2144,

(Footnote continued on next page)

Ewing,[18] in a classic statement on "General Pathology of Lymphosarcomas" summed up the matter as follows:

> . . . A search for statistical data brings to light the notable fact that there are no reliable data on this subject and also that in the present state of knowledge and the attitude of the medical public mind, there is no possibility of obtaining them . . . . From this state of affairs, one obtains support for the impression that lymphosarcoma, although a major medical problem, is a badly neglected field of observation, and that until some efforts are made to bring order into this chaos, beginning with nomenclature, little accurate information regarding the economic and social significance of this disease will be available. All we know is that lymphosarcoma is a relatively common disease, nearly always fatal, of quite obscure etiology and pathology, generally difficult of diagnosis, and little influenced by treatment. All these facts were well known to Paltauf and Kundrat fifty years ago.

Despite this state of affairs, it seems worth while to continue the reporting of interesting cases in detail which have adequate pathologic study, especially those in which unusual complications occur.

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The following report is that of a patient who had a duodenal lesion with large aneurysmal dilatation, marked stenosis of the duodenojejunal angle and evidence of perforation at the site of the tumor.

REPORT OF CASE

G. H., a white man aged 26, was admitted to Kennedy Veterans Hospital on Sept. 6, 1946, with the complaint of recurrent attacks of diffuse abdominal distention, edema of the ankles and vomiting.

History.—The history of the family was noncontributory. The patient stated that he had been entirely well until three years prior to entering the Army. At that time, there developed periumbilical pain, which usually came on about three hours after he had eaten or when the weather was hot. The pain was relieved by his taking milk and alkalis and was aggravated or precipitated by his eating fatty or fried foods. One year previously, in September 1945, while he was in the Army, the patient vomited dark brown blood for three or four days and passsed bright red blood by rectum. He was hospitalized in an Army hospital, where he received blood transfusions. He had a severe reaction from the blood transfusions and was told that he had a high titer of cold hemagglutinins. The diagnosis of peptic ulcer was made, without roentgenographic demonstration. He was returned to duty after several months in the hospital.

Lymphosarcoma of Small Intestine with Perforation, Brit. J. Surg. **26**:540-546, 1939. (*i'*) Libman, E.: Sarcoma of the Small Intestine, Am. J. M. Sc. **120**: 309-327, 1900; (*j'*) Malignant Lymphoma (Stem-Cell Type) of Jejunum, Cabot Case 26041. New England J. Med. **222**:149-152, 1940; (*k'*) Multiple Lymphosarcoma of Jejunum and Ileum, Cabot Case 28242, ibid. **226**:959-960, 1942. (*l'*) Mayo, C. W., and Robins, C. R., Jr.: Lymphosarcoma, S. Clin. North America **15**:1163-1170, 1935. (*m'*) McSwain, B., and Beal, J. M.: Lymphosarcoma of Gastrointestinal Tract: Twenty Cases, Ann. Surg. **119**:108-123, 1944. (*n'*) Menne, F. R.; Mason, D. G., and Johnston, R.: Lymphosarcoma of the Intestine: Two Cases, Arch. Surg. **45**:945-956 (Dec.) 1942. (*o'*) Minot, G. R., and Isaacs, R.: Lymphoblastoma (Malignant Lymphoma): Age and Sex Incidence, and Effect of Roentgen Ray and Radium Irradiation and Surgery, J. A. M. A. **86**: 1185-1189 (April 17); 1265-1270 (April 24) 1926. (*p'*) Moreton, R. D.: Lymphosarcoma with Primary Manifestations in the Gastrointestinal Tract: Seven Cases Studied Roentgenologically, Texas State J. Med. **41**:458-464, 1946. (*q'*) Morrison, J. E.: Tumors of Small Intestine, Brit. J. Surg. **29**:139-153, 1941. (*r'*) Morrison, W. A., and Donath, D.: Tumors of the Small Intestine, California & West. Med. **55**:235-237, 1941. (*s'*) Nickerson, D. A., and Williams, R. H.: Malignant Tumors of Small Intestine, Am. J. Path. **13**:53-64, 1937. (*t'*) Nothnagel, H., and Rolleston, H. D.: Diseases of the Intestines and Peritoneum, translated by A. Stengel, Philadelphia, W. B. Saunders Company, 1905, pp. 443-451. (*u'*) Oettle, E.: Primary Sarcoma of Small Intestine as Cause of Perforating Peritonitis, Deutsche Ztschr. f. Chir. **245**:75-79, 1935. (*v'*) Pattison, A. C.: Malignant Lymphoma of the Gastrointestinal Tract, Arch. Surg. **29**:907-922 (Dec.) 1934. (*w'*) Pissarewa, T.: Lymphogranulomatosis of the Digestive Tract, Arch. f. klin. Chir. **149**:75-91, 1927. (*x'*) Premoli, F.: Peritonitis from Perforation of Sarcoma, Rev. méd. de Rosario **21**:458-461, 1931. (*y'*) Present, A. J.: Primary Lymphosarcoma of Duodenum, Am. J. Roentgenol. **41**:545-548, 1939. (*z'*) Prey, D.; Foster, J. M., Jr., and Dennis, W.: Primary Sarcoma of Duodenum, Arch. Surg. **30**:675-684 (April) 1935. (*a''*) Ragins, A. B., and Shively, F. L., Jr.:

(Footnote continued on next page)

Although he had been placed on a dietary regimen, he continued to have occasional attacks of pain. He was discharged from the Army in December 1945, with a diagnosis of peptic ulcer. In May 1946, because of severe vomiting, pain and weakness, he was forced to give up his job of driving the local school bus. In July he noted, for the first time, swelling of the ankles and bloating of the abdomen. Because of the abdominal discomfort, he put himself on a diet consisting of little else but milk; this he consumed in large quantities. He continued to vomit occasionally. The vomitus contained partially digested food but no blood. At the beginning of the period of abdominal distention, he was having one or two bowel movements daily, and for two weeks thereafter he continued to have them. He had never had diarrhea or constipation. On the day of admission to Kennedy Hospital, he had one large bowel movement. About August 1 he had his first fainting spell, which was followed by several others. After the third attack, at his family's insistence, he entered another hopsital, on August 19. At that hospital, a Miller-Abbott tube was inserted, and the patient was given fluids, including amigen® (a hydrolysate of casein prepared by digestion with sheep and hog pancreas), plasma and dextrose. He was given penicillin intramuscularly. Two days after his admission, a flat roentgenogram of the abdomen showed gas throughout the entire gastrointestinal tract. The chest was said to be normal. The impression at that time was that of incom-

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Sarcomas of Small Intestine, Am. J. Surg. **47**:96-104, 1940. (*b''*) Raiford, T. S.: Tumors of the Small Intestine, Arch. Surg. **25**:123-177 (July); 321-355 (Aug.) 1932; (*c''*) Tumors of Small Intestine: Diagnosis, with Special Reference to X-Ray Appearance, Radiology **16**:253-270, 1931. (*d''*) Rankin, F. W.: Lympho-Sarcoma of the Small Intestines, Ann. Surg. **80**:704-711 1924. (*e''*) Rankin, F. W., and Chumley, C. L.: Lymphosarcoma of the Colon and Rectum, Minnesota Med. **12**:247-253, 1929. (*f''*) Ritter, H. H., and Shaffer, J. M.: Lymphosarcoma: Case in Which There Was an Apparent Five Year Cure, Am. J. Surg. **55**:611-612, 1942. (*g''*) Shulman, S.: Primary Lymphosarcoma of Jejunum, Am. J. Roentgenol. **46**:182-184, 1941. (*h''*) Simpson-Smith, A.: Sarcoma of the Intestine in Children, Brit. J. Surg. **26**:429-438, 1938. (*i''*) Speese, J.: Sarcoma of the Small Intestine, Ann. Surg. **59**:727-738, 1914. (*j''*) Staemmler, M.: Neoplasms of the Intestines: Two Autopsy Cases, Deutsche Ztschr. f. Chir. **46**: 296-298, 1923; (*k''*) Tumors of the Intestines, in von Bruns, P.: Deutsche Chirurgie, 1924, Stuttgart, F. Enke, no. 33a. (*l''*) Sugarbaker, E. D., and Craver, L. F.: Lymphosarcoma: A Study of One Hundred and Ninety-Six Cases with Biopsy, J. A. M. A. **115**:17 (July 6); 112 (July 13) 1940. (*m''*) Svien, H. J., and Rivers, A. B.: Lymphosarcoma Causing Obstruction at the Duodenojejunal Angle: Report of Case, Am. J. Digest. Dis. **9**:45-47 (Jan.) 1942. (*n''*) Swenson, P. C.: X-Ray Diagnosis of Primary Malignant Tumors of Small Intestine, Rev. Gastroenterol. **10**:77-91, 1943. (*o''*) Ullman, A., and Abeshouse, B. S.: Lymphosarcoma of the Small and Large Intestines, Ann. Surg. **95**:878-915, 1932. (*p''*) Usher, F. C., and Dixon, C. F.: Lymphosarcoma of the Intestines, Gastroenterology **1**:160-178, 1943. (*q''*) Weber, H. M.; Kirklin, B. R., and Pugh, D. G.: Lymphoblastoma Primary in the Gastrointestinal Tract, Am. J. Roentgenol. **48**:27-37, 1942. (*r''*) Weinstein, M. L.: Lymphosarcoma of Jejunum, Am. J. Surg. **17**:355-359, 1932. (*s''*) Weintraub, S., and Tuggle, A.: Neoplasms Involving Duodenum, Radiology **28**: 362-366, 1937. (*t''*) Whitaker, L., and Fisher, J. H.: Primary Lymphosarcoma of Ileum with Perforation and Death from Acute Peritonitis, Canad. M. A. J. **30**: 420-421, 1934. (*u''*) Winkelstein, A., and Levy, M. H.: Lymphosarcoma: Fifteen Cases; Characteristic Sigmoidoscopic Picture, Gastroenterology **1**:1093-1099, 1943.

plete obstruction of the small intestine. After a barium sulfate enema, fluoroscopic examination showed that the colon was normal but that the small bowel was distended with gas. On September 3, about two and one-half weeks later, another flat roentgenogram of the abdomen showed that the Miller-Abbott tube was coiled in the stomach, with considerable gastric secretion remaining in the stomach. The roentgenographic changes were thought to be consistent with a diagnosis of obstructing duodenal ulcer and paralytic ileus. The patient then was referred to Kennedy Veterans Hospital for definitive treatment.

Fig. 1.—Roentgenogram of abdominal cavity with patient in the anterior and upright position. Note the air-containing cavity and the fluid level. The fluid content was made up of partially digested food, which shows as a mottled opaque shadow beneath the fluid level.

Physical Examination.—The patient was observed to be somewhat cachectic and noticeably dehydrated, with extreme pallor. He was lying quietly in bed, in no apparent pain. The temperature was 98.6 F., the pulse rate 92, the respiratory rate 24 and the blood pressure 88 systolic and 55 diastolic.

Head: No tenderness was observed. The pupils reacted actively and equally to light and in accommodation. No aural tenderness or discharge was noted. There was no nasal obstruction. The mucous membranes of the oral cavity were dry, but otherwise normal.

Neck: No abnormalities were observed.

Lymph Nodes: No lymph nodes were palpable in the neck, the axillas or the epitrochlear or inguinal regions.

Chest: No tenderness was noted; expansion was limited but equal bilaterally. There was no impairment of resonance and no rales. The heart did appear enlarged on percussion; the sounds were of good quality; rhythm was normal, and there were no murmurs.

Abdomen: The abdomen was soft; it was noticeably distended, without tenderness; no unusual peristalsis was seen. Auscultation disclosed occasional

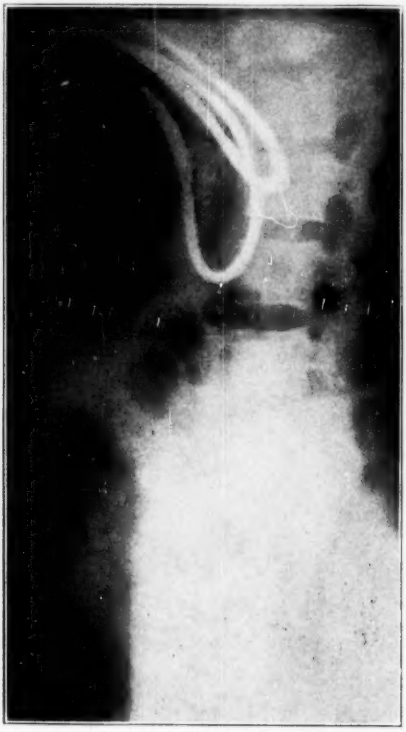

Fig. 2.—Roentgenogram of the lateral view of the abdomen taken in the upright position, showing that the Miller-Abbott tube lies in the duodenum and has not entered the aneurysmal dilatation containing air and fluid. The entire small intestinal tract is displaced against the posterior abdominal wall.

borborygmi and splash in the right lower quadrant and the umbilical region, especially in the former site. The abdomen was hyperresonant, with tympany anteriorly. There was equivocal shifting dulness. No masses were felt; the liver and spleen were not palpable.

Rectum: Examination revealed nothing abnormal. The patient had a Miller-Abott tube in place, but there was no drainage at the time of examination.

Clinical Impression.—The tentative diagnosis was subacute perforation of a peptic ulcer with subacute obstruction of the small intestine. The patient was too ill to be examined further.

Laboratory Findings (Sept. 6, 1946).—The hemogram showed 3,100,000 red cells and 7,600 white cells per cubic millimeter, with 91 per cent polymorphonuclear cells. The hemoglobin concentration was 55 per cent (Sahli). The serologic reactions were negative. Total proteins measured 6.2 mg.; albumin, 3.9 mg.; globulin, 2.3 mg.; (albumin-globulin ratio, 1.7); nonprotein nitrogen, 33 mg., and chlorides, 610 mg. per hundred cubic centimeters.

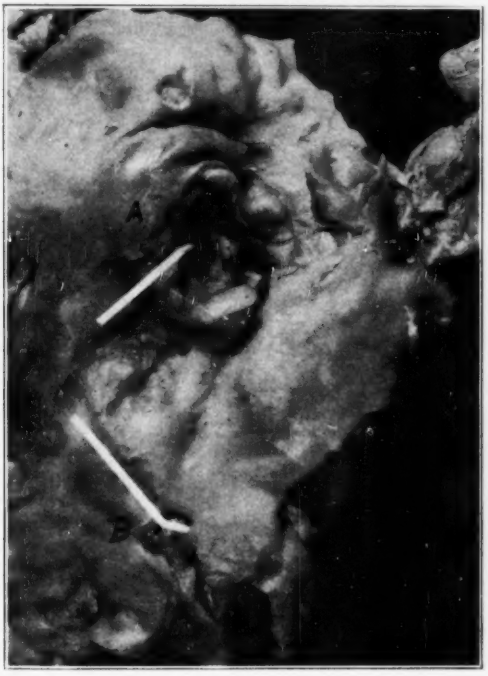

Fig. 3.—Gross specimen of the diseased portion of the terminal duodenum. Site *A* shows the orifice of the less diseased portion of the duodenum as it enters the cavity. Note the large lumen of the orifice. Site *B* reveals the orifice of the efferent portion of the bowel, which is distinctly stenosed. The stenosed area is located at the beginning of the jejunum.

Urinalysis showed a specific gravity of 1.021, a trace of albumin, innumerable hyaline casts and some granular casts.

Roentgenographic Examination.—Both sides of the diaphragm were in high position. A small air bubble was present in the fundus of the stomach. No air was shown immediately below the diaphragmatic domes. There was a slight

pleural thickening in the lower left side of the chest. No significant pathologic condition was present in the lungs.

Abdomen: A huge, walled-off cavity was present in the anterosuperior portion of the abdomen. It measured about 26 cm. in width, 23 cm. in length and 16 cm. in depth. Approximately one half was filled with fluid, with a freely shifting level. The mottled appearance of the fluid shadow was due to intermingling of the fluid with gas bubbles, and possibly to the presence of fibrin. A huge gas bubble was present above the fluid level. The roof of this cavity was at the level of the twelfth dorsal to the first lumbar vertebra. It was sealed off from the subdiaphragmatic area, probably by adherent omentum. The stomach was pressed against the posterior abdominal wall. It contained only a

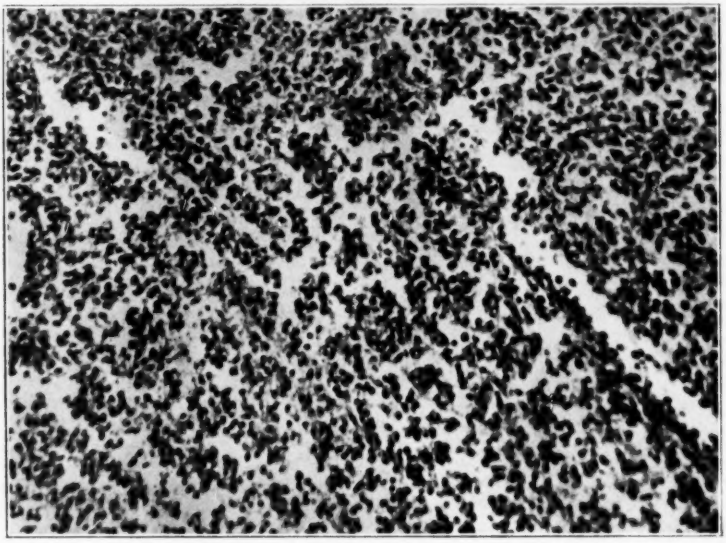

Fig. 4.—Photomicrograph of section of the tumor revealing cells of the reticular type embedded in a fine reticular stroma. The diagnosis was reticulum cell lymphosarcoma.

small gas bubble and the figure-of-eight loop of a Miller-Abbott tube, of which the terminal end extended through the pylorus and the duodenum to the vicinity of the ligament of Treitz. The entire tract of the small intestine was displaced against the posterior abdominal wall. No appreciable amount of air was contained in it, and its location could be established only by small scattered droplets of mercury coming from the first bulb of the Abbott tube. The cecum and the lower portion of the descending colon were in normal position, while the transverse colon and the flexures were displaced posteriorly. The colon was outlined by gas content, which was within normal limits. A few gas-capped, small fluid levels were present in the lower and upper abdominal areas, on the

right side, most of them situated in the colon. Intestinal stasis and distention, if present at all, appeared to be slight (figs. 1 and 2).

Opinion.—The diagnosis was that of a huge intra-abdominal abscess, containing fluid and gas, which was extravisceral, as described.

Operation.—On the morning following the patient's admission, September 7, operation was performed. When the abdominal wall was opened through an incision in the upper part of the right rectus muscle, the peritoneum was observed to be densely adherent to a mass which was unidentifiable but seemed cystic. After much difficulty, a survey of the stomach and the first part of the duodenum revealed no pathologic changes. The liver and gallbladder were normal. The right kidney was normal. The transverse colon was not involved

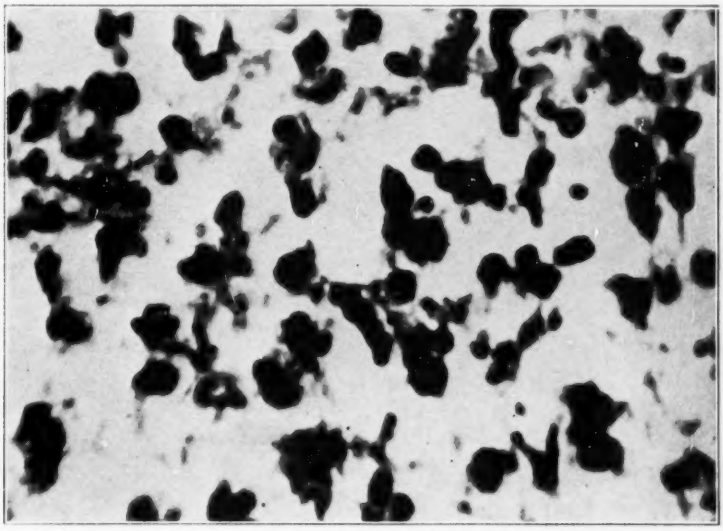

Fig. 5.—Photomicrograph with high power lens of section of the tumor especially stained to show the reticular stroma. The cells have irregular borders and thin protoplasmic projections. The cell nuclei are hyperchromatic. A rare multinucleated cell may be noted.

in the process but lay over and superior to it. The pancreas was rather indurated and firm but suggested no acute process. There was a tremendous amount of clear transudate in the peritoneal cavity outside the cystic tumor. The mass seemed to arise inferior to the transverse mesocolon and measured about 17 by 20 cm. in its greatest diameter. The superior wall of the mass seemed to include a portion of the mesocolon. The mass was opened, and over 200 cc. of partially digested food was removed. The walls of the cystic mass were thickened and friable and did not bleed freely. A brownish secretion, suggesting bile-tinged fluid, began to flow into the cavity. The specimen taken for biopsy showed granulation tissue. Four drains were inserted in the cystic cavity, and

one drain was inserted external to the cyst wall, in the free peritoneal cavity. The abdomen was then closed, using stainless steel wire.

Postoperative Clinical Course.—On the second postoperative day, the patient began to show signs of being moribund. With the use of plasma proteins, blood transfusions and dextrose solutions, his condition improved. A special suction apparatus, devised by the house staff, functioned efficiently in keeping the patient's abdomen dry. All secretion was collected and measured as an aid in determining fluid replacement needs.

The patient had improved so much that on the fifth postoperative day an enterostomy was performed through a small incision in the left rectus muscle, and a catheter was inserted for feeding the patient. On the ninth postoperative day

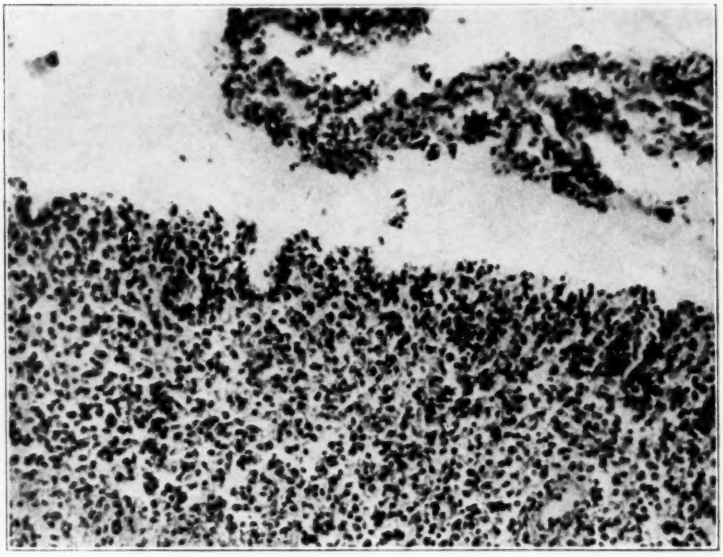

Fig. 6.—Photomicrograph showing the invasion of the jejunal wall by lymphosarcoma. This portion of the bowel adjoined the aneurysmal dilatation of the duodenum.

there was partial disruption of the incision in the right rectus muscle. The wound was repaired with through and through sutures of braided silk. At this time evidence of mild jaundice became apparent. The icteric index was 60. The urine gave a positive reaction for urobilin and urobilinogen.

Every effort was made to maintain the food intake at a level of 2,500 calories, given through the enterostomy tube and parenterally. The patient received from 200 to 300 Gr. of protein daily. Maintenance of the fluid balance was a problem, and careful checks were made periodically in order that both the fluid and the electrolyte balance might be maintained.

During his stay in the hospital, the patient's condition fluctuated from day to day. There were periods of drowsiness and listlessness which did not seem to be

correlated with the fluctuating nonprotein nitrogen of the blood. On September 22, the fifteenth day after operation, the wound again ruptured, with partial evisceration, and the condition was handled conservatively by strapping. Increasing amounts of fluid were noted in the abdomen. The abdominal cavity was tapped, and 3,500 cc. of straw-colored fluid was obtained. Peripheral edema appeared, and it was obvious that the patient was losing ground. On September 23, the sixteenth postoperative day, he became drowsy and was mentally confused at times. Bloody fluid, at first bright red and later dark brown, began to drain from the site of the cystic cavity. On September 24 his condition became grave, and on September 25, the eighteenth postoperative day, he died.

During the course of the patient's hospitalization, the true nature of the disease was not known.

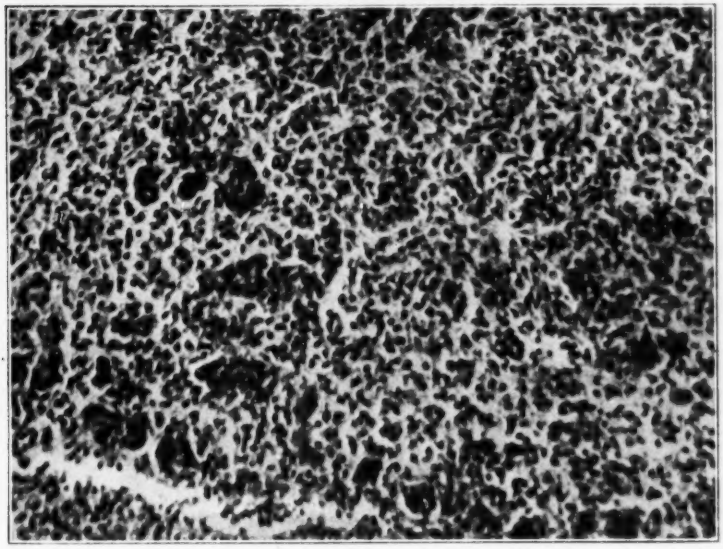

Fig. 7.—Photomicrograph of a section of the pancreas invaded by tumor tissue, which in some areas was largely replaced with lymphosarcoma.

Necropsy was performed on September 26.

Summary of Autopsy Observations.—The skin had a definite icteric discoloration. The abdomen was conspicuously distended; a midline surgical incision, 16 cm. in length, was open over its lower two thirds. There was slight edema of the lower extremities.

The abdomen, when opened, contained approximately 6,000 cc. of bile-tinged fluid. This fluid was pocketed in two areas, the upper part of the abdomen and the pelvis. In the midportion of the abdomen, there was a large space, approximately 20 cm. in diameter, which contained considerable clotted blood and which communicated with the wound in the abdominal midline. This space was lined with a thick yellow and gray inflammatory wall, composed partially of omentum

anteriorly, loops of small bowel and mesentery posteriorly and the transverse colon along the upper border. These observations indicated a walled-off perforation of the tumor. The mass was densely adherent to the anterior abdominal wall, and dissection of the intestinal tract was accomplished with difficulty. There was considerable fibrinous exudate on the surface of the small intestine and over the right dome of the liver.

On close examination, the terminal portion of the duodenum was observed to enter the left upper portion of the mass, where it opened in direct communication with the aforementioned cavity. This opening measured about 4 cm. in diameter. At a point 4 cm. below the duodenal opening a small opening, 1.2 cm. in diameter, lead from the cavity and communicated directly with the first portion of the jejunum (fig. 3). The pancreas was closely adherent to the posterior portion of the inflammatory mass over the head and the anterior part of the body. The mesenteric, retroperitoneal, preaortic and pelvic lymph nodes were enlarged.

The brain, lungs, heart, spleen, liver, pancreas, bladder and prostate gland showed no gross evidence of metastasis.

The stomach, when opened, showed nothing of note. It contained a small amount of tenacious mucus. The duodenum showed congestion and entered the inflammatory mass, as previously described. The jejunum showed nothing of note except in the lower portion, where there were several discolored, dark red areas in the mucosa. The lumen of the jejunum, ileum and colon contained a foul-smelling, dark gray, purulent material.

Anatomic Diagnosis.—The diagnosis was (*a*) destruction of the distal portion of the duodenum with formation of an aneurysmal cavity in the tumor; (*b*) perforation of the duodenum in the distal half with localized peritonitis; (*c*) lymphadenopathy (mesenteric, retroperitoneal, preaortic and inguinal); (*d*) moderate congestion of viscera, and (*e*) generalized icterus.

Microscopic Examination.—Tumor and Intestine (figs. 4, 5 and 6): Multiple sections of the tumor revealed cells of the reticular type embedded in, and separated by, a fine reticular stroma. The cells had irregular borders and thin protoplasmic projections. These were more evident with special stain. The nuclei of the cells were hyperchromatic, and many were indented or folded. Scattered multinucleated tumor cells were seen, but these were rare. The tumor replaced all normal architecture of the intestine and the lymph nodes. It was widely infiltrative.

Pancreas (fig. 7): Both acini and islets were normal in some areas. In most of the section there was infiltration by malignant tumor cells, which were irregular in outline, with single, hyperchromatic nuclei; and many of the cells showed cytoplasmic projections. A reticular stroma was present in the tumor tissue.

Microscopic Diagnosis.—The microscopic diagnosis was reticulum cell sarcoma of the duodenum, with extension to the mesenteric, retroperitoneal, preaortic and inguinal nodes and to the pancreas.

COMMENT

Lymphosarcoma is a true neoplasm arising in lymphatic tissue. It may be either a localized or a diffuse process and is distinguished from other forms of lymphoma by its local destructive capacity and by the

formation of true metastases in distant organs. Kundrat,[1g'] in 1893, first recognized it as a disease distinct from pseudoleukemia. Except for his contention that the extension was solely by permeation of lymphatic vessels, his criteria for diagnosis are still eminently satisfactory.

At first, the disease is largly confined to the lymphatic system. A chain of lymph nodes may be affected, with contiguous nodes successively involved; less frequently, widely separated groups of nodes become involved in rapid succession, or even simultaneously. The spread of the process may be lymphatic, hematogenous or by direct extension.

Sugarbaker and Craver [11n'] have classified lymphosarcoma as a process either of monocentric origin or of multicentric origin. The disease as a process of monocentric origin is limited to a localized area in the beginning and may be attacked radically by surgical measures or by irradiation. Primary intestinal lesions fall into this group. As a process of multicentric origin, multiple areas become involved simultaneously. They may spread by the lymphatics and show true metastatic growths in other organs.

Gross Pathologic Features.—Lymphosarcoma may appear grossly as (*a*) a polypoid lesion, (*b*) an infiltrating annular lesion in the intestinal wall with thickening and little evidence of constriction, (*c*) a stenosing annular lesion, usually associated with extensive infiltration of the intestinal wall, or (*d*) an aneurysmal dilatation at the site of the tumor or above it. The annular and polypoid varieties of growth may be present together. The annular type is commoner than the polypoid. The polypoid lesions frequently are multiple.[2]

In many cases there is a decided tendency to aneurysmal dilatation at the site of the tumor. This dilatation is explained on the basis of the nature of the lymphosarcoma, which, by its aggressiveness, destroys the intestinal wall; because of its lack of fibrous structure, permits dilatation of tumor structure, and, because of its infiltrative character, destroys nerve supply to the regional muscularis. Dilatation of the bowel is seen frequently above the tumor, as a result of partial or complete obstruction at the site of the lesion. Stenosis of the bowel in lymphosarcoma is seen much less frequently and, as a rule, is dependent on a fibrosis reaction (as in our case) or on compression by mesenteric lymph nodes.[3] Intussusception [4] is not a

2. Assman.[1a] Crowther.[1m] Frank, Miller and Bell.[1a] Geschickter.[1x] Present.[1y'] Raiford.[1b''] Svien and Rivers.[1m''] Ullman and Abeshouse.[1o'']

3. Fraser.[1v] Morrison.[1q'] Staemmler.[1k''] Ullman and Abeshouse.[1o''] Weinstein.[1r''] Winkelstein and Levy.[1u'']

4. Kasemeyer.[1e'] Raiford.[1b''] Ullman and Abeshouse.[1o'']

problem in cases of lymphosarcoma of the duodenum. Perforation of bowel invaded by lymphosarcoma occurs occasionally in any portion of the gastrointestinal tract. Lewis [1h'] reported 6 instances from the literature, with a case of his own. In addition to its occurrence in our case, perforation of the diseased bowel as a complication is indicated in a few other cases reported in the literature.[5] Lymphosarcoma is rarely associated with other types of tumor in the intestine.

Histopathologic Fracture.—Applying the principle of morphology as a basis for the classification of lymph nodes, Ewing [1s] designated two types of lymphosarcoma: (*a*) reticulum cell sarcoma, probably arising from the reticulum cell of the germ centers, of follicles or of pulp cords and (*b*) malignant lymphocytoma, the lymphocyte being the cell of origin.

Various classifications [6] are available for lymph nodes, some of which have considerable merit; but we feel that until the basic facts known about the tumors of lymphoid tissue are increased, the classification given here is highly satisfactory for lymphosarcoma.

Reticulum cell sarcoma is much the commoner of the two pathologic conditions. The structure of this tumor presents a diffuse growth of lymphoid cells lying in reticular tissue. The structure of the affected node or follicle is largely obliterated. The cells vary in size from small to large. The nuclei may be either compact or vesicular. Nucleoli are not prominent, but hyperchromatism is a constant feature. Large multinuclear cells are occasionally seen.

Malignant lymphocytoma presents the picture of proliferation of lymphocytes, the reticulum remaining passive. Uniformity of cells, the aggressive characteristics of the lesions and widespread metastases help to distinguish it from other forms of lymphoma, of which pseudoleukemia and lymphatic leukemia are examples.

The earliest stages of lymphosarcoma begin in the follicles of the submucosa as a local thickening of the submucosa, with or without ulceration. The process extends laterally and invades and destroys the muscularis. It may appear as a subserous tumor. The intestinal wall may become diffusely thickened and tubelike. Thereafter, central ulceration excavates the tissue or produces aneurysmal dilatation, which, with peripheral growth, yields a large tumor with a roomy cavity. Less frequently does one find marked stenosis. Polypoid lesions in the intestinal lumen are fairly frequent.

Incidence.—An extensive review of the literature compels us to admit that an accurate compilation of cases of primary lymphosarcoma

5. Cheever.[1j] Davies.[1o] Fraser.[1v] Hulbert.[1e'] Morrison.[1q'] Oettle.[1u'] Pissarewa.[1w'] Premoli.[1x'] Usher and Dixon.[1p''] Weber, Kirklin and Pugh.[1q'']

6. Callender.[1f] Gall and Mallory.[1w] Graves.[1z]

(monocentric origin) of the duodenum is impossible. Certain generalizations of value may be made. Lymphosarcoma is more frequent in the small intestine than in the large bowel, including the rectum, the ratio being approximately 2:1.

A review of the literature from 1932 to 1947 yielded the following statistics, in order of the frequency of the anatomic location. 1. Single primary lesion: ileum, 78 cases; jejunum, 29 cases; duodenum, 9 cases, and duodenojejunal angle, 4 cases. 2. Multiple involvement: duodenum and stomach, 2 cases; jejunum and ileum, 2 cases; ileocecal region, 4 cases, and "small intestine," 4 cases.

Comparison of these figures with the statistics of Ullman and Abeshouse [1o''] and others does not alter the order of frequency for the anatomic sites. Prey, Foster and Dennis,[1w'] in a review of the literature in 1935, found 61 authentic cases of primary sarcoma of the duodenum, in 34 of which the tumor was a lymphosarcoma. Comprehensive reviews of earlier authors are those of Goldstein,[1y] Crowther,[1m] Libman [1v'] and Staemmler.[1j'']

Sex: Males show a greater predisposition to the disease than do females, the ratio being approximately 3:1. The disease is more likely to develop in Caucasians than in Negroes, the ratio being 4:1.

Age Distribution: Lymphosarcoma may occur at any age but is most frequently found in the first, third and fourth decades of life. The number of patients with lymphosarcoma of the small intestine in the first decade of life is striking.[7]

Metastases.—Lymphosarcoma spreads by direct invasion, by the lymph nodes and by the hematogenous route. The growth tends to spread early to lymph nodes in the mesentery of the part of the intestine involved. This poses a serious complication in the successful surgical extirpation of the disease. In advanced cases, practically every organ has been found to be involved. Some metastases are noted in almost every case in which complete studies have been made on patients not successfully treated. The hematogenous route is necessary in accounting for many of these secondary deposits.

Symptoms and Findings.—Tumors of the small intestine do not produce a characteristic clinical syndrome; primary lymphosarcoma is no exception. The type and character of the symptoms are varied and appear to be dependent on the location of the lesion, the rapidity of growth, the degree of intestinal obstruction and other complications caused by it. There are three main clinical pictures: 1. The patient presents symptoms of vague abdominal distress, postprandial bloating,

7. Charache.[1b,1] Cutler, Stark and Scott.[1n] Lewis.[1h'] Simpson-Smith.[1h'']

pain in the epigastric or umbilical region and, frequently, various degrees of anemia and weakness. The patients are thin and cachectic, especially when the tumor has been of long duration. Bowel movements are frequently irregular. The stools occasionally contain blood or mucus. Mild constipation, alternating with diarrhea, may be a feature. Nausea and vomiting are commonly present. 2. Recurrent attacks, suggesting obstruction but clearing in two or three days, may ultimately give the picture of chronic obstruction. 3. Subacute or acute intestinal obstruction may develop either without previous difficulty or superimposed on milder abdominal distress of longer duration. The duration of symptoms may vary from a few hours to several years.

In cases of duodenal lesions the abdominal findings are not constant. Diffuse or localized tenderness is usually present. A mass is rarely felt. Abdominal distention in varying degrees may be noted. There is usually little, if any, rigidity.

Hematologic Studies.—Hemograms reveal little of value. Sugarbaker and Craver [11r] found only 10 per cent of patients with lymphosarcoma with a hemoglobin of less than 70 per cent and only 23 per cent of patients with mild leukocytosis, with a white cell count above 10,000. There is no indication that the white cell count reflected any suggestion of lymphoid leukemia. In our patient, the hemoglobin was 55 per cent and the white cell count 7,600, with 91 per cent polymorphonuclear cells.

Roentgenographic Diagnosis.—The finding of defects in the upper intestinal tract has improved in the last two decades with more accurate methods of study. There is no characteristic roentgenographic picture of lymphosarcoma in any part of the intestinal tract. Weber, Kirklin and Pugh,[19r] in an excellent discussion, pointed out that even a review of roentgenograms taken of patients known to have lymphosarcoma gave no tangible clues that diagnosis can be improved. In 19 of 25 cases, they made an unqualified diagnosis of carcinoma of the bowel; in 1 case the diagnosis was polypoid tumor, and in 3 cases the lesions were called an ulcerating malignant growth. In duodenal lesions, dilatation of the stomach and duodenum may be observed rarely; deformity of the duodenal cap, disturbance of mucosal pattern, polypoid defects and narrowing or indeterminate changes may be seen. Pressure defects of the duodenal bulb, distortion of normal duodenal swing and defects on the greater curvature of the antrum of the stomach may occasionally also be found. When the clinical picture suggests obstruction, a roentgenologic examination without the use of opaque substances may show a dilated stomach and the intestine filled with gas. The use of the Miller-Abbott tube in conjunction with roentgenographic studies has yielded helpful information. After the tube has successfully passed into the duodenum,

a thin, standard opaque mixture, which may outline a deformity or narrowing, is passed through the tube. Such a mixture can readily be withdrawn; in this way the danger of obstruction is minimized.[8]

Treatment.—The successful treatment of primary lymphosarcoma of the intestine depends on early diagnosis and the institution of prompt treatment. Unfortunately, the treatment of lymphosarcoma of the duodenum apparently has been uniformly poor. Many cases have been reported before sufficient time has elapsed to determine the outcome of the treatment. A review of the literature showed a survival rate of from a few days to forty-eight months or longer after operation for patients with lymphosarcoma of the duodenum. Many of these patients have since died, whereas others were without evidence of disease at the time of the reports. In cases of lymphosarcoma of the ileum and jejunum, survivals up to nine years without evidence of disease have been reported. The survival rate improves for patients with lesions in the lower portion of the small intestine, especially the ileum. The anatomic relationship of bowel, mesenteric nodes and retroperitoneal nodes undoubtedly plays a significant role in the prognosis. This is due to the fact that it is possible to resect some of the earlier lesions in the ileum and jejunum which may have extended to the mesenteric nodes and yet have not extended into the retroperitoneal area. In the case of the duodenum, because of its anatomic relationship to surrounding organs and the retroperitoneal nodes, it apparently has been possible only rarely to resect the diseased bowel in this area before it has extended into the contiguous organs or tissue.

Treatment may be (*a*) definitive or (*b*) palliative. Definitive treatment is directed toward the complete removal of the disease surgically. If this does not appear possible at operation, a side-tracking procedure is indicated. There is much controversy over the efficacy of irradiation in the treatment of lymphosarcoma of the intestinal tract. However, in a careful analysis of selected reports which contain adequate information, one may conclude that irradiation as a palliative measure should be used whenever possible in cases in which definitive surgical treatment cannot be accomplished, and should be carried out in cases of the operable lesions to prevent recurrence and metastasis.[9]

SUMMARY

A case of reticulum cell lymphosarcoma of the third portion of the duodenum, with aneurysmal dilatation and subacute perforation, is

8. Greenfield.[1a'] Holmes, Dresser and Camp.[1b'] Moreton.[1p'] Present.[1y'] Prey, Foster and Dennis.[1z'] Svien and Rivers.[1m''] Swenson.[1n''] Weber, Kirklin and Pugh.[1q''] Weintraub and Tuggle.[1s'']

9. Chont.[1k] Desjardins and Ford.[1p] Minot and Isaacs.[1o'] Sugarbaker and Craver.[21''] Ullman and Abeshouse.[1o'']

reported. Stenosis of the jejunum at the distal end of the tumor process and extension of the growth into the pancreas and the regional lymph nodes were additional complications.

The pathology, symptomatology, physical observations, roentgenographic findings and treatment of lymphosarcoma of the duodenum are discussed.

The literature on lymphosarcoma of the small bowel is reviewed, especially that between the years 1932 and 1947.

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BENIGN NONEPITHELIAL TUMORS OF THE STOMACH

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THE MATERIAL for this analysis was obtained from the records of the Jewish Hospital during the past twenty years and includes 20 cases of benign tumors of connective tissue and smooth muscle origin. It is of interest to note that during the same period there were observed at the hospital 11 cases of benign epithelial growths of the stomach and 9 cases of malignant nonepithelial tumors. The evidence in our material points to the fact that the benign nonepithelial growths of the stomach are twice as common as the benign epithelial growths and approximately two and a half times as common as the malignant connective tissue neoplasms. Some of the patients gave a history of gastric disturbances which may well have been due to the presence of the tumor, but none of these manifestations could be considered pathognomonic. Abdominal pain of variable character was the most common complaint. A palpable mass was an infrequent finding; it was definitely palpated in 1 instance and questionably in another. Few patients, indeed, presented clinical evidence indicative of gastric neoplasm. Many of these growths were found incidentally at autopsy or during exploratory operations for other lesions in the gastrointestinal tract.

There is no notable sex predilection for these neoplasms of the stomach, although, as for tumors in general, males may be slightly to the fore. Of the 20 patients reported on here, 13 were men and 7 were women. The majority of these tumors have been reported to occur between the ages of 30 and 40 years, but the age incidence is wide, ranging in this series between 37 and 77 years, with an average of 60 years.

There are no definite criteria to guide one to a diagnosis of these gastric neoplasms. The tumors rarely grow to a size sufficient to cause symptoms due to pressure or to interfere with the normal function of the stomach.

From the departments of laboratories and surgery, Jewish Hospital of Brooklyn.

INCIDENCE OF DIFFERENT FORMS

An outline of the different forms of the tumors described in this paper is given in the accompanying table. It is evident that leiomyoma is more frequent in occurrence than the other forms of benign tumors of the stomach; it occurred in 12 instances, or in 60 per cent of the cases. The other forms occur more rarely and are of little clinical significance except that they may grow at times to an unusually large size and cause symptoms because of pressure.

Leiomyomas.—Clinically, not all myomas of the stomach give rise to symptoms. Many of these tumors are discovered accidentally at autopsy; only few present symptoms referable to the lesion. It is stated that the outstanding clinical features of the reported cases of leiomyoma of the stomach are mechanical obstruction and gastric bleeding. Repeated episodes of melena are found in an appreciable number of instances; they were found three times in the present series. The bleeding is due

Incidence of Tumors in 20 Cases

Leiomyoma	12
Fibroma	5
Neurofibroma	1
Lipoma	2

primarily to ulceration of the surface of the tumor. The less common clinical manifestations of the lesion are pain, dyspepsia, loss of weight and symptoms resulting from secondary anemia. A mass is infrequently felt on abdominal palpation, because the tumor rarely becomes large enough to be felt. Roentgenologic examination in many instances is of assistance in the diagnosis of the gastric tumor, but this is by no means true in all cases.

In the present series of leiomyoma of the stomach only 2 patients presented symptoms referable to the lesion. One case concerned a 61 year old man who complained of having lost 25 pounds (11.3 Kg.) in weight during the previous six years. Physical examination revealed a sense of resistance just above and to the left of the umbilicus; the abdomen otherwise was essentially normal to palpation. Gastric analysis disclosed normal acidity. The stools revealed evidence of occult blood on chemical analysis. Roentgenologic examination showed a filling defect in the pyloric end of the stomach. The clinical impression was that of carcinoma or peptic ulcer of the stomach. At operation there was a nodular mass in the pylorus, 9 by 7.2 by 6.5 cm., with an ulcerated surface. On microscopic examination the tumor proved to be a leiomyoma.

The second case concerned a 53 year old man who complained of epigastric pain and constipation of several months' duration. Abdominal examination revealed nothing significant. Gastric analysis showed normal acidity; the stools contained occult blood. Roentgenologic examination of the gastrointestinal tract was noncontributory. The clinical impression was that of carcinoma of the duodenum, and at operation there was a nodular, circumscribed, firm mass in the pyloric portion of the stomach which was 4 by 2 by 2 cm. The free surface of the mass was ulcerated and bleeding. Microscopic examination of the tumor revealed a typical picture of a benign leiomyoma.

The remaining 10 tumors of leiomyoma were incidental findings at operation or at autopsy. In 1 patient a leiomyoma of the pylorus was found at operation together with a peptic ulcer in the same region. This patient, however, presented clinical evidence indicative of a peptic ulcer and not of the neoplasm. In another patient a leiomyoma of the stomach was found in association with a carcinoma of the colon. A third patient presented clinical evidence suggestive of a gastric lesion, the nature of which was not determined until he died as a result of a coronary occlusion, when at autopsy an ulcerating leiomyoma of the stomach was discovered. All the remaining patients presented no clinical manifestations of a gastric lesion, and the tumor in each instance was an incidental finding at necropsy.

Anatomically, the leiomyoma is a small, nodular growth, rarely exceeding a few centimeters in diameter. The largest tumor in this series measured 9 cm. in diameter and the smallest 1 cm. The tumor frequently arises in the muscular coats and tends to grow inwardly toward the lumen, where it may ulcerate the mucosa and cause bleeding. The most frequent location of the tumor is in the pylorus, although it may arise anywhere in the gastric wall. The tumor is often confused clinically with peptic ulcer or with carcinoma of the stomach.

The cell origin of this tumor is from the smooth muscle of the wall of the stomach. Both in cytologic detail and in architectural pattern the tumor mimics closely the smooth muscle tissue from which it arises and offers, therefore, little anatomic difficulty in diagnosis. Microscopically, it is made up of whorls and interlacing bundles of smooth muscle cells, a varying amount of fibrous supporting stroma being present. Necrosis, hyaline degeneration and hemorrhage into the tumor mass have been described but are rather infrequent findings. Only three of the tumors in this series showed ulceration of the surface and bleeding; in all the remaining cases there were no secondary changes (fig. 1).

Fibroma.—There were 5 instances of fibroma of the stomach in the present series, one associated with a gastric ulcer and another with carcinoma of the colon. In the remaining 3 cases the fibromas were

incidental findings at autopsy. None of the patients presented clinical evidence of a gastric neoplasm. The tumors were small, not exceeding 1 cm. in diameter. They were located either subserously or intramurally, and were firm, nodular and encapsulated. The cut surfaces of the tumors were of a homogeneous, gray-white appearance.

Microscopically, the tumors consisted of dense bundles of elongated cell fibers running in different directions but in an orderly fashion; the

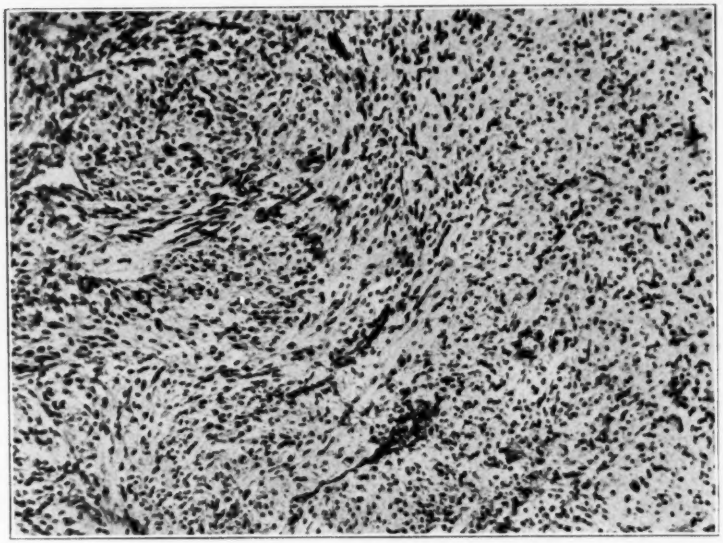

Fig. 1.—Photomicrograph of leiomyoma. Hematoxylin and eosin; × 100.

nuclei were rod or spindle shaped. Areas of dense hyaline changes, with loss of cellularity and nuclear material, were also encountered at irregular intervals. One of the tumors showed scattered areas of hemorrhage (fig. 2 *A*).

Neurofibroma.—There was only 1 instance of neurofibroma of the stomach in this series. It occurred in a 38 year old woman who complained of epigastric pain and hematemesis. Abdominal examination was essentially noncontributory aside from the presence of a palpable, nodular mass just above the umbilicus. Gastric acidity was normal; gastric extraction showed the presence of gross blood. The hemoglobin content was 34 per cent but rose to 77 per cent after several blood transfusions. Roentgenologic examination of the stomach disclosed a pocket in the pars media of the greater curvature. The clinical interpretation was that

of ulcer or carcinoma of the stomach. At operation a lobulated, moderately firm mass was found in the submucosa of the greater curvature of the stomach. A subtotal gastrectomy was performed, and the patient made an uneventful recovery.

Fig. 2.—*A*, photomicrograph of fibroma. *B*, photomicrograph of neurofibroma. Hematoxylin and eosin; × 100.

Gross examination of the specimen removed at operation revealed a nodular mass 9.5 by 8 by 5.5 cm. within the greater curvature of the stomach. The mucosa moved freely over the tumor; the latter was well

encapsulated. Cut section of the tumor showed a homogeneous, yellow-gray surface with scattered areas of necrosis and amber-colored detritus. Microscopically, it was made up of fascicles of fibers with rod-shaped or slightly oval nuclei, with typical palisade formation characteristic of neurogenic tumors (fig. 2 *B*).

Since the recognition of neurofibroma as a separate entity, there has been no uniformity of opinion as to the etiology and origin of these rare growths of the digestive tract. It is believed by some observers that the fibrous tissue of the nerve sheath is the probable site of origin. A perusal of the literature shows that there is much uncertainty and confusion in the classification of these neoplasms. Lhermitte and Leroux [1] were able to identify tumors situated in the wall of the digestive tract, the structure of which appeared in all respects to be identical with that of growths occurring in the peripheral nerves and in the spinal nerve roots. Masson [2] felt that the fundamental constituent of the tumors is the schwannian syncytium, a neurectodermal structure and not a mesodermal fibroblast.

From a review of the recent literature, it is evident that there is still a difference of opinion as to the classification and histogenesis of these tumors. A satisfactory classification is difficult, owing to the complex structure of the nerve sheath tumors and the uncertainty of the source of the supporting cells which accompany the nerve fibers and which may participate in the growth of the tumor. Masson stated the belief that the cells of the neurilemma are the parent tissue. One must consider the possibility, however, that the tumors may grow not only from the sheath of Schwann but also from the connective tissue. A careful study of several sections taken at random from various parts of the tumor in our series failed to shed any additional light on the pathogenesis. We are inclined to believe, however, that the tumor arises in all probability from the neurilemma, with which it appears closely associated.

Neurofibromas of the stomach are regularly benign, though they occasionally manifest a low degree of malignancy. The mass is encapsulated and frequently pedunculated; most of the specimens described in the literature are often ovoid or spherical. Benign tumors are apt to be harder and firmer than their malignant counterparts.

Histologically, the tumors contain whorls of interlacing bundles of fibers with a frequent palisade arrangement, a distinctive appearance peculiar to growths known as neurofibromas.

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1. Lhermitte, J., and Leroux, R.: Etude histologique générale des gliomes des nerfs périphériques des racines rachidiennes et des gliomes viscerieux, Rev. neurol. **7**:286, 1923.

2. Masson, P.: Experimental and Spontaneous Schwannomas, Am. J. Path. **8**:1, 1932.

Lipoma.—Two cases of lipoma of the stomach were noted in the present series, both occurring in persons past the age of 70 years. The tumors were incidental findings at autopsy, and the patients presented no signs or symptoms referable to them. One of the tumors was discovered in a 76 year old woman who also had an ulcer of the stomach. The other was found in a 71 year old man who died of carcinoma of the prostate. In each instance the tumor was small, soft, pedunculated and encapsulated, averaging 2 cm. in diameter. Microscopic examination of the specimens removed at autopsy revealed a typical lipomatous structure. The entire tumor was made up of closely packed polygonal cells with little supporting fibrous tissue stroma (fig. 3).

COMMENT

A review of the cases cited in this paper and those reported in the literature demonstrate clearly that the diagnosis of benign nonepithelial tumors of the stomach is often beset with many difficulties. The signs and symptoms are by themselves not distinctive and are similar to those associated with other gastric lesions. The laboratory tests, although of great value, are by no means infallible.

Analysis of the diagnostic facts relating to these tumors shows that only a small number of the patients present symptoms referable to the lesion. Many of the patients cited here were symptom free and the tumor was discovered incidentally at autopsy or during exploratory operations for other gastrointestinal lesions. The patients who presented symptoms referable to the stomach disclosed that abdominal pain was perhaps the most important single complaint; it varied from a sensation of fulness to sharp abdominal pain and was mostly referred to the epigastrium or immediately around it. Most of the symptoms were due to obstruction or to bleeding resulting from ulceration of the surface of the tumor.

Practically all the patients operated on presented past histories which may have had bearing on their illness. Some experienced transient attacks of pain which varied in duration from several weeks to several months. The patients were mostly well nourished; only 1 patient lost considerable weight. One patient appeared acutely ill on entering the hospital. Tenderness over the affected area was elicited in 2 patients. A palpable mass in the epigastrium was noted in 1 patient and questionably in another.

The laboratory data were not at all conclusive. In cases in which the tumor was ulcerated and bleeding the patients manifested signs of secondary anemia. Also, occult blood was found in the stools or free blood in the material aspirated from the stomach. Gastric analysis, whenever done, showed normal acidity. Roentgen examination was a major aid in the diagnosis of the tumors. It revealed the presence of a

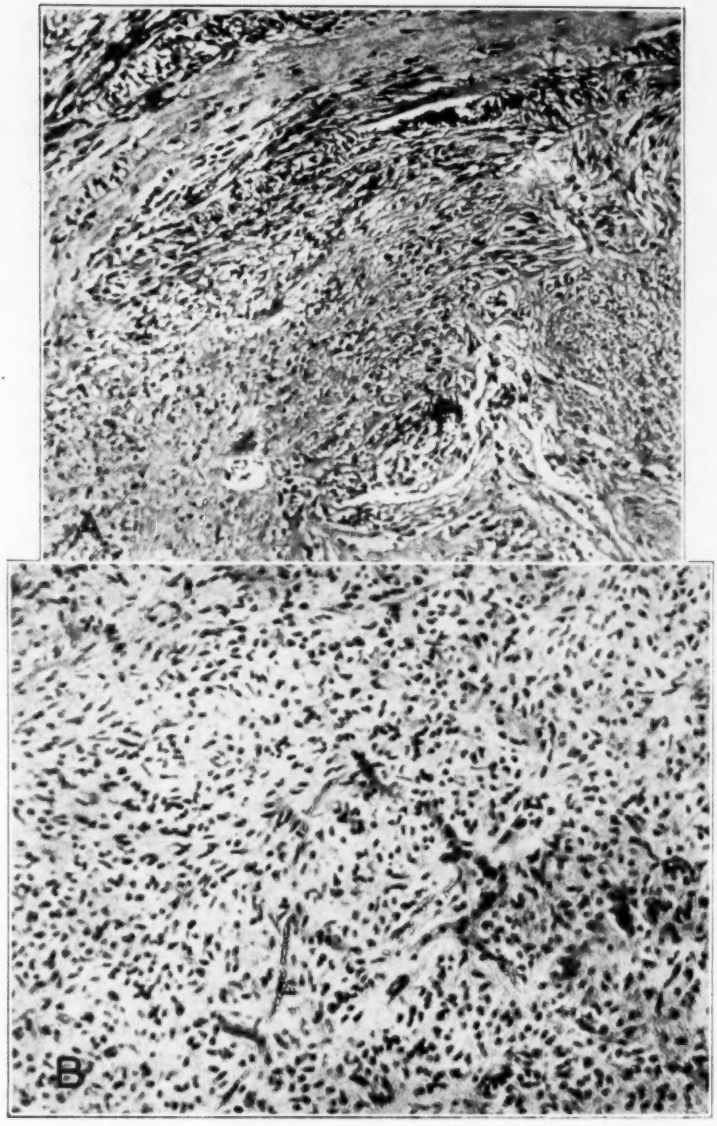

Fig. 3.—Photomicrograph of lipoma. Hematoxylin and eosin; × 20.

defect in the stomach indicative of a gastric lesion, although its true nature could not be definitely ascertained from the roentgen examination alone.

The general peculiarities shown by these patients enable the following brief summary to be made: A diagnosis of the lesion may be entertained in view of the age of the patient, the short clinical history, the normal gastric acidity, roentgen studies, maintenance of good nutrition and the presence of a palpable abdominal mass. These factors, considered singly or in combination, should lead one to suspect the lesion. This holds true particularly in the case of leiomyoma and neurofibroma of the stomach. Lipomas and fibromas, on the other hand, rarely give rise to symptoms indicative of their presence. Since myomas and neurofibromas are often associated with mucosal ulceration, bleeding is an important item in the clinical history. There were no instances in this series of emergency surgical intervention for acute massive hemorrhage as is sometimes seen in cases of gastric ulcer or carcinoma.

As has already been noted, these tumors are often closely mimicked by other gastric disturbances, notably peptic ulcer and malignant growth of the stomach. This is mainly due to the fact that they are located in the pyloric or prepyloric region, where gastric ulcer and carcinoma are most commonly found. Also, the tendency for the tumors to cause mucosal ulceration and bleeding complicates the picture still further. The tumors, however, display certain features which are lacking in both peptic ulcers and malignant growths of the stomach and which exert an important influence on the management of the disease. The general tendency for patients to maintain good nutrition and normal acidity and the short duration of symptoms at once preclude malignant tumors of the stomach. Similarly, the lack of a definite clinical history of peptic ulcer and the absence of a high gastric acidity speak well against peptic ulcer. The clinical results of operative treatment of these benign tumors of the stomach are also better than those in cases of either gastric ulcer or carcinoma.

The pathologic picture presented at operation is rather striking. The tumor usually appears as a nodular, relatively immobile, firm mass in the pyloric or prepyloric region of the stomach. It is usually confined to the muscular coats or submucosa, and the overlying mucosa is freely movable over it. Ulceration of the surface is relatively frequent and gives rise to bleeding. The tumors being benign, they are usually well encapsulated and can be enucleated.

The histologic descriptions of these tumors in the literature are similar to those in our series. It seems unnecessary, therefore, to repeat the histologic picture in detail, but a few points might be reemphasized. In the case of leiomyoma, the tumor is made up almost entirely of whorls and interlacing bundles of smooth muscle fibers enclosed within a well

formed capsule of fibrous tissue. The tumor mimics closely the mother tissue from which it arises and offers, therefore, little difficulty in diagnosis. The fibromas offer little difficulty in anatomic diagnosis. They are made up of dense collections of fibroblastic tissue which often become hyalinized and lose their cellularity and their nuclear material. The neurofibromas are of particular academic interest because of their infrequent occurrence and peculiar histologic structure. The tumor cells form whorls of interlacing bundles with a characteristic palisade arrangement, a feature which renders their histologic diagnosis simple. The lipomas show a typical microscopic picture peculiar to all fatty tumors. The growth is made up of closely placed fat cells with little connective tissue stroma to support them. These tumors in the stomach are usually small and rarely give rise to any symptoms.

The question of classification of the tumors has more than academic significance, as it bears largely on the form of treatment to be adopted. While benign tumors may be removed by simple excision, malignant tumors, on the other hand, require considerably more extensive surgical intervention to assure a cure. Choice of operation depends on the size, the variety and the situation and on the condition of the patient. The ideal treatment is complete removal if possible. Small tumors, especially when encapsulated, may be readily enucleated with little interference in gastric continuity or gastric function. When the tumor is unusually large and the question of malignancy arises, wide resection of the tumor-bearing portion of the stomach, with reestablishment of gastrointestinal continuity, may become necessary.

Little need be said of the prognosis in cases of these tumors. Since they are benign they can be removed with low mortality rate and with little fear of recurrence.

SUMMARY

Twenty cases of benign, nonepithelial tumor of the stomach are reviewed. In 12 of these the lesion was leiomyoma, in 5 fibroma, in 1 neurofibroma and in 2 lipoma.

The diagnosis of these tumors is difficult because of the similarity in signs and symptoms to other gastric lesions. The essential features presented by them are generally described.

The main histologic characteristics of the various forms of the tumors noted in the present series are described.

Surgical Forum

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From a number of questions submitted, the following were selected because of their common application to technical problems concerning gastric resection for duodenal ulcer. These questions were submitted to the editors, and the answers are summarized. It should be emphasized that this forum is meant to be informal as well as informative. Review of the literature or dogmatic answers are not its goal but rather ideas, experiences and opinions which may in turn stimulate careful and scientific investigation and thus lead to deductions of lasting therapeutic value.

Readers are invited to report their findings, whether in agreement or not, and to submit experiences as well as further questions about this or other general surgical problems.

1. In the performance of a partial gastrectomy for duodenal ulcer, is there convincing evidence that excision of the ulcer (aside from cases of massive hemorrhage) leads to better clinical results?

Only one editor deviates from an unequivocal negative answer by stating that there is slight evidence in favor of excision of the ulcer. However, the details of this evidence are difficult to obtain. One of the important reasons suggested was the difficulty in separating the cases in which the ulcer was excised from those in which it was left, even by careful examination of the specimen. Thus the surgeon may describe excision of the ulcer, yet the pathologist may fail to find it. This, however, is no proof that the ulcer was not removed, inasmuch as crushing clamps are frequently applied over the ulcer, leading to its removal from the patient yet preventing its identification by the pathologist.

Further comment emphasizes the possibility of leakage at the duodenal stump when the ulcer is broken into during the dissection. The mortality rate, after gastrectomy, improved when surgeons ceased trying to excise the duodenal ulcer in certain cases in which the procedure proved difficult. On the other hand, the removal of all antral cells down to the pylorus is extremely important.

Whether actual perforation occurs in cases in which the ulcer has been definitely left has not been reported in a large series; however, it must be unusual, although one editor describes an experience in which this accident occurred during the early postoperative period.

2. Could you refer to any follow-up information proving that the amount of stomach removed (in addition to the prepyloric region) influences the more permanent relief of symptoms?

Removal of all antral cells is a sine qua non. This is probably due to the need for eliminating the effect of these cells which seem to stimulate the secretion of acid gastric juice. Excision of the cells probably also stops the production of lysozyme, a ferment which apparently dissolves the protecting mucin normally secreted by the rest of the stomach and which thus is said to be a factor in the pathogenesis of ulcer.

Wide resection is favored by all, with emphasis on the lesser curvature by one and on the fundus by another. One editor reports an excellent follow-up study of patients operated on between 1940 and 1946, who were divided into two general groups, one subjected to "high" resection and the other to "low" resection. Proved jejunal ulcer was found in but 2 per cent of the former and 8.3 per cent of the

latter. Moreover, from the evaluation of the late results and the application of rigid standards, it was observed that the high resection gave (among those surviving operation) 81 per cent over-all satisfaction, in contrast to 71 per cent for the low resections.

One of the difficulties in answering this question stems from unavoidable inaccuracy in the use of terms. It is almost impossible to place the meanings of "wide," "high," "low," "partial" "subtotal" resection on an exact quantitative basis although many attempts have been made. Roentgenologic comparisons of preoperative and postoperative barium fill-up in patients with no gastric distention might be worth while investigating (see also Walters, W., and Taylor, E. R.: Gastric Resection for Duodenal Ulcer, Questions and Answers, Arch. Surg. **57**:697 [Nov.] 1948).

3. In the performance of a posterior gastroenterostomy after resection, does it make any difference whether the lesser curvature is placed proximal or distal to the greater curvature?

No unanimity was apparent, although there is a definite preference for the anastomosis in which the distal jejunum lies in juxtaposition with the greater curvature. Variations in this detail may depend considerably on variations in the anatomic relationships found in individual patients, emphasis being placed on avoiding any kinking of the proximal jejunum lying between the ligament of Treitz and the anastomosis. A preference is expressed for as short a loop of proximal jejunum as possible. On the other hand, the point is made that many types of anastomoses, including antecolic, with or without enteroenterostomy are described in the literature by surgeons, with apparently excellent postoperative results.

Mention should be made of the possibility of allowing the stump of the stomach to rotate as it will so that the site of both the lesser and greater curvatures may be sutured not necessarily at each end of the anastomosis but at its anterior or posterior row. Perhaps this should be investigated in a series of cases.

4. On what factors does the early restoration of oral intake after gastrectomy depend?

Careful and complete preoperative and postoperative care is stressed, with special reference to the avoidance of edema rather than to purely mechanical factors. The prevention or correction of edema depends first of all on adequate relief before operation of stagnation with its resultant inflammation in patients with pyloric obstruction. Almost as important is an adequate intake of protein to correct as much of the protein deficiency as possible by one or more of the presently available methods. A third and extremely important factor is the avoidance of excessive parenteral administration of sodium chloride solution, excessive being defined as any significant amount over and above the actual needs for replacement and maintenance.

PROGRESS IN ORTHOPEDIC SURGERY FOR 1946

A Review Prepared by an Editorial Board of the American Academy of Orthopaedic Surgeons

XIV. DISEASES OF GROWING AND ADULT BONE

Prepared by
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Growth of Bone.—Very little was added to knowledge of this subject during the year 1946. Armstrong [684] reports experimental work on rats in which one limb is completely denervated; at maturity the animal is killed, and the limbs are compared. As a result of his work, Armstrong concludes that paralysis of a limb interferes with appositional growth of bones more than with epiphysial growth, and that growth of bone is not prevented in the denervated limb.

Osteitis Deformans.—Of several reports, that of Newman [685] is most valuable. Eighty-two cases are described. In only 48 per cent did the patients have complaints referable to osteitis deformans (Paget's disease); in the remaining 52 per cent the disease was an incidental finding. The pelvis was involved in the majority of cases, and in 12 only one bony structure was affected. Serum calcium and phosphorus determinations revealed little variation from normal. The level of alkaline phosphatase was elevated in 6 of 9 cases in which the examination was made; this test is considered by the author to be the most useful laboratory procedure in current use in establishing the diagnosis of Paget's disease. Complications observed in the series, considered in order of their frequency, were (1) pressure on the cranial nerves, (2) fractures, (3) urinary calculi and (4) sarcomatous degeneration. Magnesium carbonate U. S. P. produced symptomatic improvement in 6 of 8 cases in which it was tried; its use constitutes the only therapy of value.

Summey and Pressly [686] give an excellent review of the clinical and microscopic picture of Paget's disease. The various possible etiologic

684. Armstrong, W. D.: Bone Growth in Paralyzed Limbs, Proc. Soc. Exper. Biol. & Med. **61**:358-362 (April) 1946.

685. Newman, F. W.: Paget's Disease: A Statistical Study of Eighty-Two Cases, J. Bone & Joint Surg. **28**:798-804 (Oct.) 1946.

686. Summey, T. J., and Pressly, C. L.: Sarcoma Complicating Paget's Disease, Ann. Surg. **123**:135-153 (Jan.) 1946.

factors are discussed. The value of the paper lies chiefly in the attempt of the authors to record all reported cases of sarcoma complicating Paget's disease; these number 76. The incidence of sarcoma in several reported series of cases varies from 2 to 14 per cent. In reported cases the femur, humerus, skull and tibia, in that order, are the bones most frequently involved. The authors report 3 cases of their own in detail, and state the belief that amputation is the treatment of choice where possible, considering the gravity of the prognosis.

Moehlig and Abbott [687] call attention to the close relation of disordered carbohydrate metabolism to osteoporosis and Paget's disease. Studies of the blood were made in 94 cases of osteoporosis and in 40 cases of Paget's disease. Glucose tolerance tests revealed a diabetic type of curve in 35 of 40 cases of osteoporosis and in 27 of 31 cases of Paget's disease. The authors are of the opinion that since arteriosclerosis almost invariably accompanies both conditions, the administration of calcium and vitamin D is contraindicated.

In an interesting and thought-provoking article, Edholm, Howarth and McMichael [688] present data which indicate that in generalized Paget's disease there occurs an increase in the blood flow in the bone of sufficient magnitude to produce effects on the general circulation similar to those resulting from free arteriovenous communications.

Fibrous Dysplasia of Bone.—Interest in the fibrous conditions of bone continues and will continue for some time to come, and there is still diversity of opinion. Murray, Kirkpatrick and Forrai [689] report a case of Albright's syndrome. It is of particular interest in that it occurred in a male patient who presented progressive fibrous dysplasia, roentgenologic changes and absence of changes in the blood suggesting parathyroid adenoma and of pigmentation of the skin. Pronounced clinical deformity of the bones of the lower extremities was present and prevented ambulation. The patient was made ambulatory with braces by excision of segments of bone, so as to prevent stress on vessels and nerves at the time of correction of the deformities.

Patten [690] reports an incomplete form of Albright's syndrome without precocious puberty and pigmentation of the skin. The total number

687. Moehlig, R. C., and Abbott, H. L.: Carbohydrate Metabolism in Osteoporosis and Paget's Disease, J. Michigan M. Soc. **45**:642-646 (May) 1946.

688. Edholm, O. G.; Howarth, S., and McMichael, J.: Heart Failure and Bone Blood Flow in Osteitis Deformans, Clin. Sc. **5**:249-260 (Dec.) 1945.

689. Murray, R. C.; Kirkpatrick, H. J. R., and Forrai, E.: A Case of Albright's Syndrome (Osteitis Fibrosa Disseminata), Brit. J. Surg. **34**:48-57 (July) 1946.

690. Patten, R. F.: Polyostotic Fibrous Dysplasia (Albright's Syndrome), Clin. Proc. Child. Hosp. **2**:175-184 (July) 1946.

of cases is thus 59, 39 of which are of the complete and 20 of the incomplete form. They occurred in the proportion of 3 female to 2 male patients.

Hatcher [691] presents a thoughtful and outstanding paper. He does not accept as bone tumors per se the localized benign fibrous lesions of bone which Jaffe and Lichenstein classified as benign tumors originating from connective tissue of matured marrow. The basis for the report is a study of 45 patients, with 51 lesions. The disorder has its inception during the period of longitudinal growth, for all but 7 of the patients were children. In all patients the lower extremities were affected, and in 5 patients more than one bone was involved. Symptoms are usually mild; in a number of cases the fibrous areas were discovered accidentally. The roentgenographic appearance of the lesion is distinctive and reveals an area of reduced density in the metaphysis close to the epiphysial disk. Later the area is apt to be elongated and is usually eccentrically placed within the bone. Sharp demarcation from surrounding normal bone by a narrow shell of bone sclerosis is usually observed. Irregular scalloped margins give the appearance of loculation. Through longitudinal growth, the defect comes to lie farther from the epiphysial cartilage, and in the process of tubulation of the metaphysis the defect may eventually occupy a part of the cortex. Surgical removal was done in 17 cases. Though the pathologic appearance varies somewhat, the basis in all stages is a fibrous connective tissue which occupies a smooth-walled, and sometimes partially loculated, cavity in the bone. The newly formed focus shows a relatively cellular fibrous tissue, with scattered multinucleated cells. In what appear to be older foci, the fibrous tissue is arranged in strands and whorls. Lipid-filled macrophages are often present and are believed to be a sign of chronicity. It was evident in the cases under observation that in many instances the lesion became obliterated spontaneously by the reparative ossification of tubulation of the bone. In the author's opinion the association of the metaphysial fibrous pockets with disorders of epiphysial bone and cartilage is too frequent to be mere coincidence. With the exception of Legg-Calvé-Perthes disease (osteochondrosis of the capital epiphysis of the femur), the incidence of fibrous defects corresponds well with the localization of epiphysial disorders. While no clue as to the primary causation of the disturbance is available, the association with demonstrable epiphysial disorders is

691. Hatcher, C. H.: Pathogenesis of Localized Fibrous Lesions in the Metaphyses of Long Bones, Ann. Surg. **122**:1016-1030 (Dec.) 1945.

considered significant. The author believes that the association is further borne out by a similarity in course, which suggests that the fibrous lesion of the metaphysis may also follow vascular derangement.

Gaucher's Disease.—Moore [692] presents 2 cases of this rather rare condition, which is probably due to a little understood derangement of lipid metabolism. The author states the belief that splenectomy does not alter the course of the disease as regards bone, and reports that roentgen therapy failed to give relief.

Rickets.—An interesting report on nonrachitic bowlegs in childhood may be noted. Leonard and Cohen [693] emphasize the nonrachitic nature of bowlegs occurring as a result of osteochondrosis deformans of the tibia. A unique plan of nonsurgical treatment, used in three stages, is presented for treatment of the infantile type of this condition: (1) immobilization of the limbs in plaster until the bones soften; (2) gradual correction of the deformity by wedging casts, and (3) resumption of activity, with the protection of braces until the abnormal growth process becomes static. Convincing tracings of the progress of correction in a number of cases are presented.

Infarction of Bone.—Kahlstrom and Phemister [694] describe the results of autopsy in a case previously reported. The material is presented after careful and critical study; excellent photographs give gross, microscopic and roentgenoscopic details of massive lesions of both femurs and one tibia. The actual cause of the infarction was undetermined but was thought to be related to a generalized and coronary arteriosclerosis. The authors state the belief that infarcts in the condition are present much more frequently than they are found.

Since the bends (caisson disease) at times causes infarctions of bone, the report of Burkhardt and associates [695] is included. They attempted to ascertain whether the use of gas, visible roentgenoscopically, might elucidate the cause of the bends. Seven susceptible subjects were repeatedly exposed to an altitude of 38,000 feet (11,600 meters), and four hundred roentgenograms were made. In only 22 instances was

692. Moore, M., Jr.: Bone Lesions in Gaucher's Disease, Memphis M. J. **21**:132-136 (Sept.) 1946.

693. Leonard, D. W., and Cohen, L.: Nonrachitic Bowlegs in Childhood: Osteochondrosis Deformans Tibiae, J. Pediat. **29**:477-484 (Oct.) 1946.

694. Kahlstrom, S. C., and Phemister, D. B.: Bone Infarcts, Am. J. Path. **22**:947-963 (Sept.) 1946.

695. Burkhardt, W. L., and others: A Roentgenographic Study of "Bends" and "Chokes" at Altitude, J. Aviation Med. **17**:462-477 (Oct.) 1946.

a roentgenographic picture characteristic of bends obtained. Pain was not related to the amount of gas found in joint cavities, bursae or vaginal sheaths. Extra-articular collections of gas were located as frequently anterior to the femur and knee joint as posterior to them. A correlation between the pain and the amount of gas, as measured on the lateral roentgenograms, was not found; pain was always present, however, when the area of gas measured exceeded 0.9 sq. in. (5.8 sq. cm.). The authors conclude that production of pain by a bubble or bubbles depends on the chance location of the bubble at a point of extra-articular tissue where nerves or nerve endings are readily distorted.

Osteosclerosis.—Kelley and Lawiah [696] present 4 cases of this disease in one generation of a family. There was no evidence of the condition in the two previous generations, but 4 of 10 children were affected. The condition is of unknown cause, is familial and is characterized by an increase in the roentgenologic density of bones, with preservation of their structural contour. It is usually widespread throughout the skeletal system. Progressive anemia results from destruction of bone marrow, and the cranial nerves are injured by encroachment of bone on the foramens.

Ehlers-Danlos Syndrome.—Holt [697] reports the cases of 2 sisters having this rare condition, which is ordinarily characterized by (1) hyperelasticity and fragility of the skin and blood vessels, (2) hypermobility of the joints, (3) pseudotumors over the bony prominences and (4) movable nodules beneath the skin. The roentgenograms are particularly interesting, as they reveal disseminated, small, rounded, discrete, calcareous nodules. These consist of lobules of calcified necrotic fat, surrounded by dense fibrous tissue capsules, which the author believes to be due to repeated trauma.

King-Lewis [698] reports briefly 2 additional cases.

Eccentro-osteochondrodysplasia (Morquio's Disease).—This skeletal disease is characterized by (1) dwarfism, (2) deformities of the bones of the trunk and extremities and (3) roentgenologic changes, consisting of absence of ossification centers and destruction, rarefaction and proliferation of all the bones of the body, including the skull. Two phases of the disease, namely, structural defects and neurologic changes,

696. Kelley, C. H., and Lawiah, J. W.: Albers-Schoenberg Disease, a Family Survey, Radiology **47**:507-513 (Nov.) 1946.

697. Holt, J. F.: The Ehlers-Danlos Syndrome, Am. J. Roentgenol. **55**:420-423 (April) 1946.

698. King-Lewis, F. L.: Ehlers-Danlos Syndrome: Two Cases, Proc. Roy. Soc. Med. **39**:135 (Jan.) 1946.

are described in a report by Einhorn, Moore and Rowntree.[699] They also describe the only reported autopsy in a case of this disease. An abnormal relation was noted between the first cervical segment and the skull, causing pronounced compression of the brain stem and providing an excellent explanation of the neuromuscular changes observed in the condition.

Scott and Rotondo [700] report a third case in a Negro child.

Calcification.—Howard [701] reports 5 cases of "traumatic ossifying myositis," or ossifying hematoma. The condition in these instances followed football and soccer injuries in Naval cadets taking preflight training. The author believes that the frequency of this complication of football injuries may not be generally appreciated and suggests that roentgen therapy may be helpful in these cases.

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XV. TUBERCULOSIS OF BONES AND JOINTS

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WILKINSON [702] discusses the relation of multiple tuberculous lesions to prognosis in the treatment of tuberculosis of bones and joints. He states the belief that associated pulmonary lesions are most important, skeletal lesions of least significance and genitourinary lesions of intermediate importance. He regards multiple infection of lymph nodes as indicating good resistance and multiple hematogenous lesions the reverse. The author feels that general benefit is gained in many cases by operation; this result may be due to removal of a source of tuberculous toxemia or to autoinoculation.

From the New York Orthopaedic Dispensary and Hospital.

699. Einhorn, N. H.; Moore, J. R., and Rowntree, L. G.: Osteochondrodystrophia Deformans (Morquio's Disease): Observations at Autopsy in One Case, Am. J. Dis. Child. **72**:536-544 (Nov.) 1946.

700. Scott, E. P., and Rotondo, C. C.: Chondro-Osteodystrophy (Morquio's Disease), Arch. Pediat. **63**:261-265 (June) 1946.

701. Howard, C.: Traumatic Ossifying Myositis, U. S. Nav. M. Bull. **46**:724-730 (May) 1946.

702. Wilkinson, M. C.: Treatment of Bone Tuberculosis in Relation to Multiple Tuberculous Lesions, Proc. Roy. Soc. Med. **39**:712-713 (Sept.) 1946.

The histories in 129 cases of skeletal tuberculosis complicated with associated tuberculous lesions, reported over a ten year period, are examined. The study shows that results in the surgical cases were slightly better than those in the nonsurgical cases, and would indicate that operation should not be withheld because of multiplicity of lesions.

[ED. NOTE (A. DeF. S.).—This evidence confirms the findings of Cleveland in a series of cases recorded at Sea View Hospital. The prognosis was affected adversely by the presence of active pulmonary tuberculosis, especially when associated with cavities and a positive sputum. However, as Wilkinson points out, some of the patients recovered, and the prognosis in this group should not be considered hopeless.]

Eriksen [703] reports on 234 proved cases of tuberculosis of the foot and ankle, in 136 of which there were active or inactive tuberculous lesions in other organs of the body. The ankle joint was involved in 50.8 per cent of the cases, and the lesion occurred in isolated tarsal bones in 14.7 per cent. Fistulas or abscesses developed in 54 per cent of cases during the course of treatment.

Conservative measures were employed until repeated roentgenoscopic examinations revealed the tuberculous process to be at a standstill. In cases of isolated foci simple evacuation of the focus was done. Other surgical procedures were resection of joints and excision of isolated bones. There were 22 amputations. Seven patients died while the foot disorder was acute, and 19 died later, in most cases as a consequence of pulmonary tuberculosis. Drop foot developed in 18 cases, and in many the destructive processes resulted in flattening of the foot or in other deformities.

[ED. NOTE.—One wonders whether the results in these cases might not have been better if surgical treatment had not been delayed until the lesions appeared to be inactive.]

Ruppanner,[704] in discussing secondary infection of tuberculous abscesses, cites a case of perforation of an abscess into the esophagus, followed by severe secondary infection, which led to erosion of the aorta, with fatal hemorrhage. He mentions 2 cases in which secondary infection of a tuberculous abscess occurred by metastasis. A young man

703. Eriksen, B.: Tuberculosis of Bones and Joints, Including Analysis of Material of Danish Disablement Pension Board of Years 1921-1940, Acta orthop. Scandinav. **16**:43-46, 1945.

704. Ruppanner, E.: Metastatische Infektion eines tuberkulösen Senkungsabszesses mit dem Bacillus funduliformis, Schweiz. med. Wchnschr. **75**:1089-1090 (Dec. 8) 1945.

with involvement of the third lumbar vertebra experienced a severe episode of influenza, after which bilateral iliofemoral abscesses became secondarily infected. Streptococci were demonstrated by culture. The patient died a few days after excision of the abscesses. In another case of tuberculosis of the eighth dorsal vertebra, an abscess developed in both the right and the left lumbar region. Puncture of the abscesses disclosed fetid, brownish pus and gram-negative bodies. Cultures revealed no bacterial growth. The abscess in the left lumbar region was later evacuated, and Bacillus funduliformis was noted in the pus. The incision failed to heal, and the patient died of severe secondary anemia.

Kisch [705] reviews 253 cases of tuberculosis in which the patients were treated at the University Institute for Joint and Bone Tuberculosis, Hohenlychen, Germany, from 1920 to 1929. The author describes surgical and conservative treatment of tuberculosis of the knee joint. Since tuberculosis of joints is a metastatic process, only the metastatic lesion can be removed surgically; the patient is not so cured, cure being possible only by treatment of the entire body, resulting in healing of the primary focus. In many cases, however, conservative treatment must be supplemented by surgical measures. The author's recommended treatment includes the use of (1) weight-bearing braces (but no plaster casts); (2) traction to prevent cramps and deformities; (3) passive motion within the limits of pain, as soon as generalized pain and spasm disappear; (4) surgical measures, such as wide excision or amputation to save the life of the patient; (5) treatment of the entire body with sun and fresh air; (6) induction of local passive congestion by Bier's rubber bandage method and local roentgen therapy, and (7) aspiration of abscesses, as indicated.

In discussing his results, the author classifies the varieties of the disease as osseous, hydrops and fungous. At the time of discharge from the hospital 103 of 163 patients with the osseous type were considered cured. Sixty-two of 79 patients with the fungous type and 9 of the 12 patients with the hydrops type were cured. The follow-up results (up to eleven years) showed that 71 of 92 patients with the osseous type had healed lesions, 15 had relapsed, 3 had improved and 3 had died. In 40 of 48 patients with the fungus type and in 9 of 12 patients with the hydrops variety the disease remained healed.

[ED. NOTE.—It should be pointed out that no proof is offered of the correctness of the diagnosis in these cases and that no follow-up study

705. Kisch, E.: Treatment of Tuberculous Arthritis, Am. Rev. Tuberc. **53**: 533-546 (June) 1946.

is detailed. The evidence accumulated over a period of thirty-five or more years on the advantages of surgical treatment is ignored.]

Herdner and Malgras [706] report 2 cases of localized tuberculosis of the costotransverse joints. The standard roentgenogram did not reveal the lesions, but by serial topography arthritis of the involved joints was demonstrated. The lesions were treated by excision of the transverse apophysis and of a portion of the articulating rib with use of local anesthesia. Both patients recovered without complications.

[ED. NOTE.—At the New York Orthopaedic Dispensary and Hospital the laminagraph has proved very helpful in demonstrating obscure lesions of the bones and joints.]

Ellonen [707] reports on the histologic examination of 104 pairs of palatine tonsils. Among 74 children with tuberculosis of the bones and joints or involvement of lymph nodes, tonsillar tuberculosis was demonstrated in 20 (27 per cent). In apparently healthy children tuberculous foci in hypertrophied tonsils are probably rare. If their presence is suspected, and the reaction to the tuberculin test is positive or the child is susceptible to infections they should be removed. In children with involvement of bones and joints tuberculous changes are not the primary cause of swelling of the tonsils, but children with a lymphatic diathesis are more subject to recurrent tonsillitis, and tubercle bacilli probably have more chance to propagate on the affected tissues.

The author states the belief that insufficient attention is paid to tuberculosis of the tonsils because signs of the disease in the tonsils are usually lacking.

[ED. NOTE.—Of the tonsils removed from 100 patients with tuberculosis of bones and joints, those of 25 were proved histologically to be tuberculous.]

Tuberculous Rheumatism.—Seligson [708] reviews the report by Poncet and Leriche and articles by several other authors who have sought to establish tuberculous rheumatism as an entity. The observations of Poncet and Leriche have not been generally accepted in the

706. Herdner and Malgras, P.: Une localisation inédite de tuberculose ostéo-articulaire, celle de l'articulation costo-transversaire, Mém. Acad. de chir. **71**:454-457 (Nov. 28-Dec. 19) 1945.

707. Ellonen, A.: Untersuchungen über die Tonsillentuberkulose mit besonderer Berücksichtigung ihrer Beziehung zu der Knochen- und Gelenktuberkulose der Kinder, Acta oto-laryng. (supp.) **47**:1-129, 1942.

708. Seligson, F.: Poncet's Disease: Clinical Observations in Inflammatory and Degenerative Joint Reactions in Tuberculosis, Am. Rev. Tuberc. **52**:463-473 (Dec.) 1945.

United States because of too many unsupported assertions. Poncet stated the belief that tuberculous toxins caused acute rheumatism in 20 per cent of patients with tuberculosis and acute serositis in 15 per cent of those with acute tuberculous rheumatism. Chronic tuberculous rheumatism is also due to these toxins. Poncet presented as proof the simultaneous or alternating occurrence of polyarthritis and tuberculosis, the occurrence of rheumatic arthritis with a fungous tumor, similarity in cytologic features of rheumatic and tuberculous effusions, rheumatic pains after injection of tuberculin and occasionally positive reactions in guinea pig tests. Caurmont and Dor produced serous arthritis by injecting attenuated tubercle bacilli intravenously. The paper emphasizes transitory inflammatory changes in joints and serous cavities, occurring with the spread of tuberculosis elsewhere in the body. Eight cases are reported.

Sheldon [709] states that tuberculous rheumatism is rare but is reported from time to time. Diagnosis may be made when rheumatic phenomena are preceded by, or accompanied with, active tuberculosis. The author refers to three clinical types described by Poncet and Leriche: arthralgia, acute rheumatism with pericarditis and chronic rheumatism. Clinically the manifestations in the joints are milder than in rheumatic fever, and there is no response to salicylates. Six cases are reported; all patients had tuberculosis, and 2 died. No attempt was made to recover acid-fast bacilli from the joints. The author concludes that tuberculous rheumatism is an expression of altered immune mechanism in a susceptible host.

Desmeules, Rousseau and Richard [710] review the theories and conclusions of Poncet's Lyons school and others and report 3 cases of their own. Two patients had pulmonary lesions; in 1 the lungs showed no evidence of involvement. All had multiple arthritis. In the 2 patients with pathologic involvement of the lung the tuberculous factor in the involvement of the joints was determined by elimination of other causes. Tubercle bacilli were demonstrated in fluid taken from the knee joint of the third patent.

Paraplegia in Spinal Tuberculosis.—Seddon [711] states that early onset of paraplegia is usually caused by a tuberculous abscess. In the thoracic region, factors which favor involvement of the spinal cord are narrowness of the spinal canal; the anterior concavity of the vertebral

709. Sheldon, W.: Tuberculous Rheumatism, Lancet **1**:119-121 (Jan. 26) 1946.

710. Desmeules, R.; Rousseau, L., and Richard P.: Rhumatisme et tuberculose, Laval méd. **11**:35-46 (Jan.) 1946.

711. Seddon, H. J.: The Pathology of Pott's Paraplegia, Proc. Roy. Soc. Med. **39**:723-730 (Sept.) 1946.

column, which encourages angulation at the site of disease, squeezing inflammatory products toward the cord, and the close confinement of the abscess by the anterior ligament. In rare cases the disease begins in the neural arch, compressing the cord from behind. Pathologic dislocation at the site of disease and incautious laminectomy, which may cause collapse of the spine and retropulsion of a tuberculous sequestrum, are listed as mechanical causes of paraplegia. When the onset is late in the course of the disease, the paraplegia is due not to stretching of the cord over a bony prominence, but, as Butler has pointed out, to reactivation of a small tuberculous mass inside the cord. The causal relation of tuberculous pachymeningitis to paraplegia has not been established. Acute paraplegia may occur without compression of the cord and is thought to be due to localized edema, secondary to abscess formation. A rapidly fatal tetraplegia developed in 1 case when the vertebral artery, lying in contact with an abscess, became thrombosed.

Alexander [712] discusses the surgical management of spinal tuberculosis. Skull traction is used in treatment of cervical conditions and in those affecting the upper part of the thorax. The patients are placed in anterior and posterior plaster molds, into which head traction is incorporated. While the patient is in traction, any necessary surgical procedure may be carried out. The author does not favor laminectomy in cases of typical tuberculosis of the thoracic part of the spine. With progressive neurologic deterioration in spite of immobilization, surgical drainage must be established. This is accomplished through a lateral approach with excision of the transverse processes and portions of the ribs. The pedicles are excised to reveal the lesion, and as much diseased material as is necessary to relieve pressure is removed. Thirteen patients were treated by this method; 3 died and 10 had complete neurologic recovery. The author warns against overlooking tuberculosis in patients who have symptoms of rupture of a low lumbar intervertebral disc. Two cases are mentioned.

Deery [713] in discussing the treatment of paraplegia due to tuberculosis of the spine (Pott's paraplegia), states that it is generally agreed that 90 per cent of patients can recover through appropriate orthopedic care; the remaining 10 per cent are considered hopeless. Laminectomy as a method of treatment of this condition has been held in disrepute in the past, owing to the high mortality rate. It should

712. Alexander, G. L.: Neurological Complications of Spinal Tuberculosis, Proc. Roy. Soc. Med. **39**:730-734 (Sept.) 1946.

713. Deery, E. M.: Laminectomy for Pott's Paraplegia, Ann. Surg. **124**:201-203 (Aug.) 1946.

carry no greater risk than laminectomy for tumor of the spinal cord or for herniation of a nucleus pulposus. Laminectomy has also been criticized as mechanically weakening the back, but this should not be an objection, for the spine, if not fused by previous operation or disease, can be fused at the time the laminectomy is done.

At the New York Orthopaedic Hospital, 23 patients have had laminectomy for Pott's paraplegia. Fifteen patients showed manometric spinal block with an elevated level of protein in the cerebrospinal fluid. Six had a high protein level without block, and 1 had no lumbar puncture. Of the 23 patients, surgical fusion had been done in 15, and spontaneous fusion had developed in 1 before laminectomy. In the remaining 7 spinal fusion was performed at the time of laminectomy.

The results in 8 patients were good; the condition of 3 was improved, and that of 10 was unimproved. The follow-up period ranged from a few months to eleven years. No deaths resulted from surgical shock. There was 1 death from tuberculous meningitis and another from generalized visceral tuberculosis. These occurred several weeks after laminectomy. Of the 23 patients, 15 were adults, for 3 of whom the results were good. In 14 patients the paraplegia was of early onset. There appears to be no relation between the type of paraplegia and the result of laminectomy. The location for laminectomy was determined by the sensory level alone, but actually the positions of the sensory level, the intraspinal pathologic state and the apex of the kyphos all corresponded closely.

Pathologic Considerations.—Orell and Hellstadius [714] describe a case of tuberculous spondylitis in which death resulted from miliary tuberculosis. A close study of the morbid changes was made at necropsy. The investigation of these authors confirmed the observations of others that secondary anterior and posterior spondylitis usually follows a primary tuberculous process in the vertebral body. They deny that shadows appearing in the vertebral body on roentgenologic examination represent atrophy, as is maintained by some writers. The case also revealed that even extensive posterior secondary spondylitis may not be demonstrable with roentgenograms; the authors comment that this is frequently the situation in cases of centrally located foci in whch the cortex is not broken. The authors state that sometimes the focus causes extensive sclerosis and osteoblastic reaction in the rest of the

714. Orell, S., and Hellstadius, A.: Interesting Case of Tuberculous Spondylitis, Nord. med. (Hygiea) **13**:670-673 (Feb. 28) 1942.

vertebral body and that this may help to mask a central area of tuberculous destruction.

Farina and Negri [715] present a case of what they term an exceedingly rare form of tuberculous spondylitis. A man aged 31 had polyperiostitis, which was quickly cured. Two years later he experienced indefinite symptoms of pulmonary disease, supposed to be tuberculous, with asthenia, anorexia, profuse sweating and emaciation, followed by lumbar fistulas, which emitted a purulent discharge. The patient died one year later. Necropsy showed spondyloperiostitis from the sixth cervical to the second lumbar vertebra, paralumbar abscesses, multiple fistulas in the lumbar region and circumscribed external pachymeningitis. No pulmonary tuberculosis was present. Histologic examination demonstrated the presence of tuberculous granulomatous tissue in the walls of the two lumbar abscesses and in the specimens from the areas of lumbar pachymeningitis. There was a subchronic inflammatory erosion of the anterior and lateral surfaces of the vertebrae and the interosseous ligaments from the sixth cervical to the first lumbar vertebra. The authors state the belief that this case is one of tuberculous spondylitis of the type described by Mandelstamm in 1933. The condition is characterized by periosteo-osteitic lesions, upward spread of the disease and a rapidly fatal course. No effective treatment is known.

Schleicher [716] presents data derived from autopsy material and from terminal and known cases of pulmonary and generalized miliary tuberculosis. Tuberculosis affects the bone marrow frequently enough to allow early detection of miliary tuberculosis by means of sternal aspiration and sectioning of gross units of the sternal marrow. The possibility is suggested that the procedure may be a useful tool in diagnosis and prognosis in the field of tuberculosis.

Differential Diagnosis.—Guri [717] discusses the problem of the differential diagnosis of pyogenic osteomyelitis of the spine with special reference to tuberculosis. He describes four clinical syndromes which may be due to the spinal lesions. There may be pain in the hip, with no clinical evidence of involvement of the hip other than limitation of

715. Farina, C., and Negri, M.: La spondilite di Mandelstamm, Ann. ital. di chir. **21**:567-580 (Sept.-Oct.) 1942.

716. Schleicher, E. M.: Miliary Tuberculosis of Bone Marrow, Am. Rev. Tuberc. **53**:115-121 (Feb.) 1946.

717. Guri, J. P.: Pyogenic Osteomyelitis of Spine: Differential Diagnosis Through Clinical and Roentgenographic Observations, J. Bone & Joint Surg. **28**:29-39 (Jan.) 1946.

extension of the thigh. Irritation of the intercostal nerves may suggest a localized abdominal condition. Meningeal symptoms may be due to intradural infection or to irritaton of the meninges by an extradural lesion. Examination of the spinal fiuid is helpful in ruling out the possibility of intradural infection. Two syndromes with pain in the back are noted: an acute form, with sudden, severe pain, in which the patient is in a toxic condition, and a subacute form, in which the condition is less toxic. A pyogenic focus elsewhere in the body may precede the pain in the back. The formation of an epidural abscess, with positive neurologic signs, may occur. If the onset is insidious, the patient may have sharp pain in the back, unrelieved by rest, and muscular spasm may be noted objectively, whereas there may be little general reaction. The author believes that the condition is probably not tuberculous when the neural arches are invaded by the infection, but states that there are no roentgenographic findings characteristic of the different types of infectious spondylitis. In the localized form of osteomyelitis an area of destruction may be surrounded by sclerosis and active bone formation. Progressive thinning of the interspace, absence of early sclerosis and minimal bone or spur formation are indicative of tuberculosis. In the diffuse pyogenic forms there are an early decrease in density and a fogginess of the surfaces of the bodies with early reactive bone formation and intense sclerosis. In the tuberculous forms pronounced atrophy occurs, with collapse of the vertebrae. Reactive bone formation is rare.

Lewis[718] describes the differential diagnosis of tuberculosis and suppurative arthritis from the point of view of the roentgenologist. His article is based on the diagnostic points set forth by Phemister in 1924. In nontuberculous pyogenic infections of the joints of the extremities, acute osteoporosis occurs, with an early decrease in the width of the joint. The earliest destruction of bone appears on the weight-bearing portion of the articular surface, and there is a tendency to repair and ankylosis. In tuberculosis there is less osteoporosis, with late persistence of the width of the joint, and the earliest destruction of bone is at the periphery of the joint. Atrophy of the muscles is usually pronounced; there is little tendency to repair or ankylosis. The author states that these factors appear not to apply to tuberculosis of the hip joint or to caries sicca. He also warns that many conditions

718. Lewis, R. W.: Differential Diagnosis of Tuberculosis in Joints of Extremities, Rev. radiol. y fisiotherap. **13**:115-122 and 164 (July-Aug.) 1946.

may simulate tuberculosis of joints. Several illustrative histories and roentgenograms are presented.

[ED. NOTE.—It is my experience that thinning of the joint space frequently is an early finding in cases of tuberculosis of the joint, although it does not progress so rapidly as in pyogenic infections. This change apparently is due to a trophic alteration in the hyaline cartilage, since this tissue is not directly invaded or destroyed by the tubercle bacillus.]

Ferey and Sambron [719] report 2 cases of tuberculosis of the spine in which the history and findings were typical of herniation of a nucleus pulposus. A 40 year old farmer, with no history of previous illness, had felt a suddent violent pain at the base of the spine when he made a false move while unloading hay. Examination three months later revealed slight atrophy of the right leg and diminution in strength of the right achilles tendon reflex. On myelographic examination the flow of iodized oil U. S. P. (lipiodol®) was seen to have stopped at the junction of the third and the fourth lumbar vertebra. Exploration revealed a small, hard tumor anterior to the dura at the level of the third and fourth lumbar vertebrae. There was no evidence of inflammatory reaction, but the tumor contained pus. The cavity was evacuated, filled and closed with a plug of muscle. The patient made an uneventful recovery.

The second case was that of a woman aged 30, who had felt a sudden, severe pain in the lower part of the back, accompanied with a cracking sound, when she lifted a bucket of water. The pain radiated to the left leg. A myelogram taken fourteen months after injury revealed the blocking of the flow of lipiodol® at the junction of the third and the fourth lumbar vertebra. There were atrophy of the left leg and weakness of the achilles tendon reflex on the left, but no sensory disturbances. Exploration disclosed a small swelling, containing pus and caseous material, at the level of the third and fourth lumbar vertebrae. The condition was treated in the same manner as that in the first case, with complete recovery.

The authors mention an earlier case, in which operation was followed by death from tuberculous meningitis. In this case, after aspiration of the contents of the abscess, followed by curettement, they applied a thin layer of muscle instead of completely filling and plugging the cavity with muscle.

719. Ferey, D., and Sambron: Deux observations de fausses hernies discales dues à des abcès froids, Rev. neurol. **77**:189-192 (July-Aug.) 1945.

[ED. NOTE.—A similar case has come to my attention, in which the patient died of tuberculous meningitis after the abscess had been drained.]

Richart Sotes [720] presents tables and gives the results of his own version of transplantation and inoculation in guinea pigs, in a report of 10 cases of suspected osteoarthritic tuberculosis. A specimen of suspected material (articular capsule or cartilage) is obtained aseptically at the time of operation and divided into three parts: one for histologic examination, one (made into a pulp) for subcutaneous injection and one for transplantation, through a buttonhole incision, into the peritoneal cavity of the guinea pig.

Tuberculosis was suspected clinically in the 10 cases, but its presence was confirmed in only 4. The method of transplantation gave positive results in all 4 cases, whereas inoculation by subcutaneous injection gave a positive result in only 1 case; histologic examination gave positive evidence in 2 cases, being of less diagnostic value than transplantation, but superior to inoculation. The author feels that his method offers a means of earlier diagnosis in cases of suspected tuberculosis. He observes that some animals died shortly after inoculation and some did not, but that in all animals caseation of the mesenteric lymph nodes occurred without affecting the serosa or becoming generalized. He offers no explanation as to why the infection takes this course.

[ED. NOTE.—Growth of tubercle bacilli on culture mediums often provides a more rapid means of diagnosis than guinea pig inoculation and is worthy of addition to the routine methods.]

Antibiotics.—Frank [721] expresses the opinion that local injection of penicillin into the fistulous tract is of distinct advantage in bringing about a permanent closure of most tuberculous fistulas with secondary infection, provided (1) the tuberculous process is no longer active, (2) the sequestrums are all removed and (3) the invading organisms are sensitive to penicillin. The author instilled penicillin into the sinus tracts twice daily, injecting under a fair degree of pressure in order to make the drug penetrate to the deepest portion of the fistula. The author states that systemic treatment with penicillin is indicated in cases of extensive fistula formation with toxic symptoms, poor

720. Richart Sotes, T.: Contribución al diagnóstico diferencial de la tuberculosis osteoarticular, Rev. españ. de tuberc. **15**:261-274 (April) 1946.

721. Frank, P.: Penicillin Therapy of Secondary Infected Abscesses, Nederl. tijdschr. v. geneesk. **90**:366-367 (April 27) 1946.

general condition of the patient and beginning amyloidosis. He further states the belief that caseous masses or poorly nourished granulation tissue in the depths of wounds should be removed before the local treatment is begun.

Four cases are presented; in 3 the fistulas closed completely in from one to four months of treatment. In the fourth case, the infection, caused by Escherichia coli, did not respond to penicillin therapy.

Lapierre [722] presents 4 cases of multiple tuberculous fistulas with secondary infection. The ages of the patients ranged from 3 to 28 years. In 3 cases lesions involving the hip and in 1 a lesion of the knee were treated by débridement of the fistulous tracts and by administration of penicillin, both locally and systemically. Laboratory tests showed gram-negative cocci, Staphylococcus albus and Staphylococcus aureus in all 4 cases; in only 1 case was Mycobacterium tuberculosis demonstrated. From the clinical course and manifestations, the condition in all 4 cases was thought to be undoubtedly of tuberculous origin. All patients were in poor general condition. Improvement was noted one week to ten days after the beginning of treatment; after the course of penicillin therapy, in which total amounts of the drug varied from 1,175,000 to 5,160,000 units, all fistulas had either closed or almost closed, with little residual discharge.

The author describes the surgical and the conservative treatment of tuberculous wounds in the orthopedic cases in the Sacré-Coeur Hospital in Montreal. When surgical procedures are planned, penicillin is given preoperatively for two or three days. The abscesses or fistulas are débrided widely and closed over rubber tubes, for use in lavage and local instillation of penicillin. Local and intramuscular administration of penicillin is continued for at least ten to twenty days after the disappearance of the agents of secondary infection. Laboratory examination of the pus is made at regular intervals.

The conservative method is used when there are too many fistulas for débridement and in cases of large atonic wounds. For such wounds the surrounding skin is washed daily with medicinal soft soap U. S. P., cleansed with ether or benzene, swabbed with a 70 per cent solution of alcohol and covered with a large dressing. Penicillin is given intramuscularly. After the secondary infection has been overcome, regeneration is encouraged with a commercial pomade called herisan,® which contains halibut liver oil, boric acid, talc, zinc peroxide and sulfathiazole.

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722. Lapierre, V.: Therapy of Secondary Infected Fistulas, with Special Reference to Penicillin, Union méd. du Canada **75**:799-784 (July) 1946.

[ED. NOTE.—Streptomycin has been remarkably successful in promoting the closure of tuberculous sinuses, presumably because of its effect on the tubercle bacilli. Possibly a combination of streptomycin and penicillin would offer the best treatment of this condition.]

Morgan and Bosworth [723] report the case of a 35 year old physician who had tuberculosis of the lung, the cervical and dorsal portions of the spine and the ilium, with draining sinuses in the neck. Treatment consisted of transfusions, the administration of 32,000,000 units of penicillin and spinal fusion, performed in two stages, from the occiput to the tenth thoracic vertebra. At this point a paraspinal abscess ruptured through the pleura and lung into the bronchial tree, and the patient's condition became critical. Streptomycin was given as a last resort, 4,000,000 units being administered over a period of twenty-four hours. The dose was then maintained at 1,000,000 units daily for fifty days. At the end of that time, the patient's general condition was greatly improved. The sinuses in the neck, which had been draining for thirteen months, closed in twenty-one days. The temperature became normal; the sedimentation rate fell to within normal limits; the sputum became negative for tubercle bacilli; coughing and raising of sputum ceased, and infiltration of the chest cleared. The author states the opinion that recovery in this case can be attributed to the administration of streptomycin.

Keefer and others,[724] in an article on streptomycin in the treatment in 1,000 cases of infection, mention 4 cases of tuberculosis of bones and joints, in 2 of which improvement was noted.

Operative Procedures in Tuberculosis of Bones and Joints.—Resection of the knee was performed by Stanischeff [725] on 53 patients who had had tuberculosis of the knee joint for an average period of eight years. Most of the patients had been treated by conservative methods for years before coming to him and had fibrous and osteofibrous ankylosis in flexion or, more rarely, in recurvation, with or without subluxation of the joint. One third of the patients had sequestrums and fistulas.

The treatment consisted of complete resection of the joint, including the patella and capsule. Any sequestrum was removed, the wound

723. Morgan, C., and Bosworth, D. S.: Transpleural Rupture of Tuberculous Spinal Abscess Treated Successfully by Streptomycin: Report of Case, J. Bone & Joint Surg. **28**:864-868 (Oct.) 1946.

724. Keefer, C. S.; Blake, F. G.; Lockwood, J. S.; Long, P. H.; Marshall, E. K., and Wood, W. B., Jr.: Streptomycin in the Treatment of Infections, J. A. M. A. **132**:4-11 (Sept.) 1946.

725. Stanischeff, A.: Knee-Resection of Joint, Minerva med. **1**:25-28 (Jan. 6) 1942.

closed and a plaster cast applied. In 42 of the 53 patients no sinuses remained after operation, and walking on the limb was satisfactory and painless.

[ED. NOTE.—My experience has been that attempts to eradicate the diseased tissue as completely as possible, by radical excision of the joint, are unnecessary and cause needless mutilation. The majority of patients recover after simple arthrodesis with minimal resection of bone.]

Servelle [726] discusses an operation for tuberculosis of the shoulder, used first by Leriche during World War I in the treatment of wounds and applied in the treatment of tuberculosis in 1930. The operation is described in detail; its chief features are meticulous excision of the fungous lesions, cauterization and curettage if necessary, and the filling of the cavity with osteoperiosteal grafts, obtained previously from the tibia. Leriche used this method in 20 cases of humeroglenoid tuberculosis in adolescents and adults. All the operative wounds healed by first intention, and in all cases ankylosis occurred rapidly. Follow-up examinations at one, two and seven years showed cure in all but 1 case, and fusion was obtained in this case after a second operation. The author states that ankylosis is obtained in two to three months by this method.

Freiberg [727] performed several Brittain ischiofemoral arthrodeses over a period of seven months in 1944; he then waited six months to determine the results in these cases, prior to repeating the procedure. Although numerous additional operations were done, studies of end results are reported only in the first 7 cases. The follow-up period was thirteen to twenty months. Six patients, from 7 to 18 years of age, had tuberculosis of the hip; the seventh patient had degenerative arthritis. In 4 of the 6 patients with tuberculous hip, osseous fusion (determined clinically and roentgenographically) resulted within seven to nine months; in 1 patient, a 7 year old girl who broke the spica several times after operation, firm fibrous union developed. She was ambulatory and asymptomatic sixteen months after operation. Renal tuberculosis developed in a 72 year old woman one month after operation, necessitating removal of the spica for nephrectomy. One year after the Brittain fusion, her hip had minimal motion clinically, and

726. Servelle: De la résection de l'épaule pour tuberculose par le procédé de Leriche, Rev. d'orthop. **32**:189-193 (May-Aug.) 1946.

727. Freiberg, J. A.: Brittain Ischiofemoral Arthrodesis, J. Bone & Joint Surg. **28**:501-512 (July) 1946.

roentgenograms indicated apparent healing. The 1 nontuberculous patient showed clinical and roentgenographic fusion within four months.

In tuberculous patients surgical procedures have not been carried out in the presence of active abscesses. Contraindications to this operation exist in those few instances in which diseased bone cannot be "short-circuited" by the osteotomy and grafting.

Bosworth and Green [728] report on fusion of the hip in 32 cases of unilateral tuberculous infection of the hip joint. Four types of surgical procedures were used: (1) the standard Hibbs technic; (2) massive transplantation of the anterior portion of the crest of the ilium, across the joint line; (3) packing with bone chips after curettage, and (4) simple curettage of the joint. Fusion was achieved in 80 per cent of cases after the Hibbs type of graft, in 70 per cent after massive inlay grafting and in 50 per cent after curettage and the addition of chips; in 1 acute case, in which treatment was by curettage, union did not take place. The average time required for solid union after the Hibbs procedure was twelve months; after massive inlay grafting it was eighteen months.

Karken [729] states that fusion of the hip in patients with tuberculosis should not be attempted before the age of 8 to 12 years because the greater trochanter is not sufficiently ossified before that time and deformities may develop. Successful fusion does not necessarily effect cure of the tuberculous process, but the chance for the disease to remain arrested is better in the fused hip than in one that has a little mobility. An opening into a tuberculous focus does not constitute an obstacle to primary union, but sinuses are more likely to develop if the process is exposed. The type of fusion should fit the case. If a short time has elapsed since the active phase of the disease, a paraarticular arthrodesis is indicated because of the better chance of avoiding the infected area. Fusion should be done in the phase of recovery, three to six years after the onset; sinuses and fistulas develop more frequently when the operation is done too early. Immobilization should be continued until roentgenograms show that the graft has thickened and is recalcifying; this process usually requires six to seven months after operation. Karken analyzes fusion of the hip in 23 cases of tuberculosis.

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728. Bosworth, D. M., and Green, L. A.: Experiences with Arthrodesis for Tuberculosis of Hip, Quart. Bull., Sea View Hosp. **8**:39-46 (Jan.) 1946.

729. Karken, A.: Arthrodesis in Treatment of Tuberculous Coxitis Sequelae. Acta orthop. Scandinav. **16**:1-27, 1945.

XVI. INFANTILE PARALYSIS

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ARMSTRONG [730] states the opinion that paralysis of an extremity beginning at an early age frequently results in disturbance of bone growth. Several experimental studies on dogs are mentioned; in these it was indicated that with growth the bone of a paralyzed limb continues to gain in mass, but at a rate lower than normal. He cites several experiments by other investigators, using rats and dogs in which the brachial plexus had been severed. Shortening of bone and atrophy occurred in all the experimental animals, to varied degrees. Armstrong's own experiment on large rats, also performed after the brachial plexus had been severed, indicated that (1) the growth in length of the radius was affected earlier, and to a greater degree, than that of the humerus; (2) the reduction of growth in diameter of the humerus was greater than the reduction of growth in length; (3) the growth in diameter of bone was reduced, but not prevented; (4) the quantity of bone and bone salt was reduced 25 per cent, and (5) the humerus, but not the radius, of both normal and paralyzed limbs of rats 132 days old practically ceased to grow in length. The author thus concludes that paralysis of a limb interferes more with appositional than with epiphysial growth, and that growth of bone is not completely prevented in a denervated limb.

Bayley [731] presents a method and subject data for predicting adult height, using data from skeletal roentgenograms and the height of the subject at the time of examination. Chronologic age must be considered when the growth of a child in the younger age group is retarded or accelerated in a considerable degree. Mention is made of the work published by Gill and Abbott, who used percentile groups according to heights at successive ages and predicted adult height from skeletal age, disregarding chronologic age if a child was retarded or accelerated more than 6 months in skeletal age. The data presented are valuable when used with roentgenograms showing ossification centers of the hand and roentgenographic evidence of other centers which

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730. Armstrong, W. D.: Bone Growth in Paralyzed Limbs, Proc. Soc. Exper. Biol. & Med. **61**:358-362 (April) 1946.

731. Bayley, N.: Tables for Predicting Adult Height from Skeletal Age and Present Height, J. Pediat. **28**:49-64 (Jan.) 1946.

have been assigned skeletal ages in predicting the adult height of children whose growth is incomplete.

Teleroentgenography, though allowing accurate records of comparative length, produces an increasing degree of magnification and distortion as the extremities grow. A method designated as orthoroentgenography is described [732]; this allows a single roentgenographic film (14 by 42 inches, or 35 by 105 cm.) to be used with three exposures in sequence, the central ray being directed successively over each of the three joints. The results of this method satisfy the following requirements, set up by the Harvard Infantile Paralysis Commission:

1. Measurement of bones should be sufficiently accurate that comparative true lengths may be recorded at any one examination.

2. The precision of measurement should remain constant for varying bone lengths in order that the true increments of growth may be computed accurately.

3. Sufficient detail should be delineated to allow detection of abnormalities in the bones and visualization of the epiphysial lines.

Hodgen and Frantz [733] present a summary of the various technics for epiphysial arrest, discuss the calculation of the optimum time for the arrest, give a table of growth expectancies and describe the methods of roentgenologic determination of leg lengths. They present the technic of the operation, a modification of the Phemister technic, using either the bone block–turning method or the Blount method, with chrome nickel stainless steel staples. An analysis of 21 cases in which arrests were performed after the method of Phemister showed good results in 48 per cent, fair results in 47 per cent and poor results in 5 per cent.

A survey of the literature on epiphysial arrest for equalization of leg length is presented,[734] with reports of deformities resulting from epiphysiodesis of the femur, the fibula and the upper end of the tibia. The authors add reports of 4 cases of deformities about the knees, 3 cases of genu valgum and 1 case of genu varum, in all of which the condition developed from two months to two and one-half years after arrest of the lower femoral and the upper tibial and fibular epiphyses. In 3 cases the deformities about the knee were sufficiently

732. Green, W. T.; Wyatt, G. M., and Anderson, M.: Orthoroentgenography as Method of Measuring Bones of Lower Extremities, J. Bone & Joint Surg. **28**:60-65 (Jan.) 1946.

733. Hodgen, J. T., and Frantz, C. H.: Arrest of Growth of Epiphyses, Arch. Surg. **53**:664-674 (Dec.) 1946.

734. Regan, J. M., and Chatterton, C. C.: Deformities Following Surgical Epiphysial Arrest, J. Bone & Joint Surg. **28**:265-272 (April) 1946.

severe to necessitate wedge-shaped osteotomies of either the upper part of the tibia or the lower part of the femur. In all the cases the deformities could be attributed to the failure of part of an epiphysis to fuse after operation for epiphysial arrest.

The basis of a paper by Fancher [735] is the experimental treatment with glandular extracts of 67 adolescent patients who were growing too rapidly. Testosterone propionate U. S. P., diethylstilbestrol U. S. P., thyroid U. S. P., calcium compounds, vitamin D and various extracts of ovary and testis were used over a period of fifteen years. (The author's own previous experimental work had shown that a sufficiently high dose of androgens or estrogens will inhibit skeletal growth in the rat.) The diagnosis in all cases was hyperpituitarism (anterior lobe). Fifty-two of the 67 patients showed an inhibition of growth (6 of these were not growing at all); the other 15 either showed no decrease or had accelerated growth as a result of the treatment. The average time of treatment was eighteen months. Treatment continued until closure of the epiphyses, when possible.

No serious ill effects were encountered. Fancher concludes that the evidence indicates that too rapid growth in children may be safely retarded with diethylstilbestrol U. S. P. or estradiol benzoate U. S. P., with the addition of calcium and vitamin D.

Napier [736] presents a splint which is designed both to maintain the thumb in a position of function when there is weakness of the thenar muscles and, at the same time, to exercise the antagonists. Essentially, the device consists of a pad placed between the thumb and the index finger and three leather rings, one each around the thumb, the index finger and the little finger. Elastic is used for traction.

[Ed. Note (C. E. I.).—Photographs shows the thumb in abduction, but not rotated internally or pulled sufficiently far across the palm. The fifth finger has a tendency to flex and abduct before the thumb moves.]

Olmos [737] describes the creation of an anterior bone block in the ankle, according to the technic of Putti, to correct deformity of the calcaneus after paralysis of the triceps surae muscle in cases of poliomyelitis. He states that many pathologic phenomena occur after paralysis of the gastrocnemius and soleus muscles. There are loss of plantar flexion, progressive elongation of the triceps surae, loss of balance between the plantar muscles and the posterior calf muscles,

735. Fancher, J. K.: Control of Stature, J. M. A. Georgia **35**:27-33 (Feb.) 1946.

736. Napier, J. R.: Corrective Splint for Paralysis of Thenar Muscles, Brit. M. J. **1**:15 (Jan. 5) 1946.

737. Olmos, V. S.: Treatment of Paralytic Calcaneus, J. Bone & Joint Surg. **28**:780-786 (Oct.) 1946.

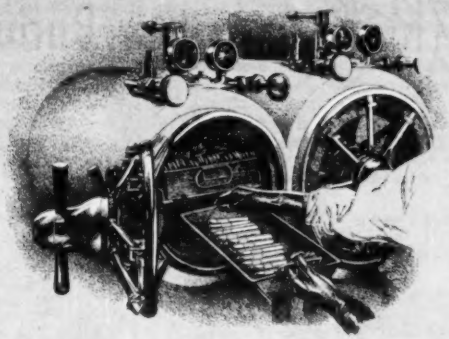
increase in the talocalcaneal angle to values above 50 to 55 degrees and structural and trophic changes in the talus, the os calcis and the investing soft tissues. There also develops, depending on the power of the remaining musculature, either cavus, when the peroneal muscles are paralyzed, or flatfoot, when the anterior and posterior tibial muscles are no longer functional. Since the power of the triceps is approximately three times the power of the muscles of dorsiflexion, tendon transplantation cannot be expected to prevent the deformity.

The operative procedure consists of a bone block built up on the neck of the talus, using cancellous bone obtained while performing the subtalar arthrodesis; the foot is then immobilized for three months in plaster, in the position of talipes equinus. The author reports good results in approximately 76 per cent of 67 cases followed for more than one year.

[ED. NOTE.—Olmos does not state at what age bone operations were done on children. No mention of plantar fasciotomy is made; this procedure is necessary in the majority of cases of true deformity of the calcaneus. When good musculature is available for transplantation to the calcaneus, the anterior bone block is not necessary. In patients with weak musculature a dangle foot is present, and fusion of the ankle should be considered if there is enough strength about the knee to remove the brace.]

CORRECTION

In the article by Drs. Smathers and Weed entitled "Treatment of Tetanus," in the September issue (ARCH. SURG. **57**:291, 1948), "mg." was used in several places instead of "units." This error appeared in the section on "D-Tubocurarine in Wax and Oil" on pages 295, 296 and 297.



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